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REFLECTION EFFECT IN ECLIPSING BINARIES FOR A POINT-SOURCE OF LIGHT

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Milne,[1] it is well known, was the first to formulate a darkening law for the reflected radiation and deduce the reflection effect in eclipsing binaries. Milne's phase law is, however, true to the order of the square of the radius (in units of the distance separating the two stars) of the reflecting component, as he considered the components to be so widely separated that the incident light constituted an approximately parallel beam.

Kopal[2] drew attention to the need for the evaluation of the reflection effect to higher order terms, as the fractional radii of the components in eclipsing binary systems are quantities of the order of one-tenth. In Kopal's words,

"In order to establish a closer picture of conditions to be met in actual binary systems..., the convergence of the cone of incident radiation should be considered as well as the finite angular size of the illuminating star. The respective exact equations belong to the class of those which are as easy to formulate as they are difficult to solve; so that relatively little is known about the behaviour of higher order reflection terms so far."

In the present paper, the finite radius of the reflecting star and the convergence of the incident beam are taken into account, but the illuminating star is treated as a luminous point.[3] The phase-function has been found correct to the fourth power of the stellar radius (in units of the distance between the two stars). The results should be applicable to supergiant components with early-type companions in eclipsing systems like ζ Aurigae or VV Cephei. The present investigation is only a first step towards a complete solution, and it is hoped that the finite radius and the darkening of the illuminating star will be considered in a future communication.

To simplify the treatment, a cosine law of reflection has been assumed, analogous to Lambert's law in planetary photometry. The results, it

may be noted, would not be greatly vitiated by this assumption, as Kopal has shown by Fourier expansion that the simple cosine law does not deviate much from the more accurate phase laws.[4] The approximation fails at the edge where, according to the cosine law, the intensity should vanish. Near the edge, however, the reflected radiation is so feeble in intensity that it contributes little to the total reflected light.[5]

We have taken as the unit of length the distance between the centers of gravity of the two stars, and have treated the reflecting star as spherical. This is valid to our order of approximation (fourth power of stellar radius), as the distortion is of the order of the cube of the radius and would therefore introduce terms of the order of the fifth and higher powers of the radius, in the reflected light.[6]

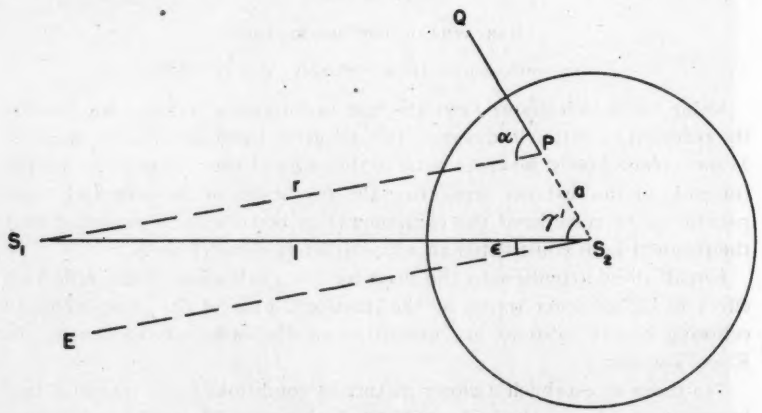

FIGURE 1

In figure 1, S_1 is the illuminating star, regarded as a point source of light. The illuminated star S_2 is of radius a, and the distance $S_1S_2 = 1$. S_2E is the direction of the observer. P is any point on the surface of S_2, and $S_1P = r$. Let the phase-angle $S_1S_2E = \epsilon$, the angle of foreshortening $PS_2E = \gamma$, the angle of incidence $S_1PQ = \alpha$, and the angle $S_1S_2P = \gamma'$.

We have, by projection on S_2Q,

$$\cos \gamma' = a + r \cos \alpha, \tag{1}$$

which gives

$$\begin{aligned} \cos \alpha &= \frac{\cos \gamma' - a}{r} \\ &= (\mu - a)(1 + P_1 a + P_2 a^2 + \ldots) \\ &= \mu - (1 - \mu^2)a - \frac{3}{2}\mu(1 - \mu^2)a^2 \ldots, \end{aligned} \tag{2}$$

where $\mu = \cos \gamma'$, and $P_1, P_2, \ldots$ are Legendre functions of μ.

We shall retain, as already mentioned, terms up to a^4.

We express the position of P by spherical polar coordinates (Fig. 2) on the sphere S_2.

N is the pole of S_1E. Let $NP = \eta$, and the angle $PNS_1 = \phi'$.

The element on the surface of S_2,

$$d\sigma = a^2 \sin \eta \, d\eta \, d\phi'. \tag{3}$$

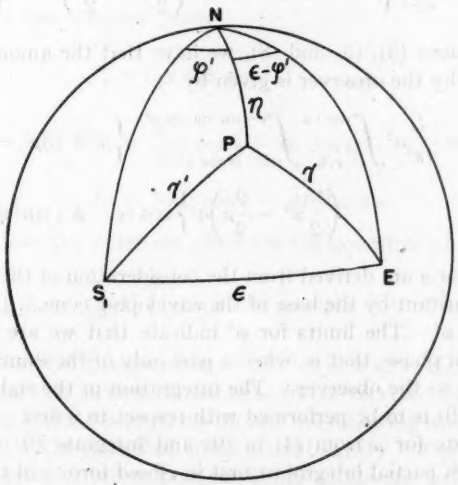

FIGURE 2

Also, from spherical triangles, NPS_1 and NPE, we have

$$\mu = \cos \gamma' = \sin \eta \cos \phi', \tag{4}$$

and

$$\cos \gamma = \sin \eta \cos (\epsilon - \phi'). \tag{5}$$

We shall assume, as already stated, that the reflected radiation is uniformly distributed in intensity according to the cosine law

$$I = S \cos \alpha, \tag{6}$$

where πS is the flux per unit area normal to itself, and α is the angle of incidence with the surface normal.

Now, we have

$$\pi S = \frac{L}{4\pi r^2}$$

$$= \frac{L}{4\pi}\{1 + 2\mu a + (4\mu^2 - 1)a^2 \ldots\}, \quad (7)$$

where L is the luminosity of the illuminating star (S_1).

From equations (2), (6) and (7), we have

$$I = \frac{L}{4\pi^2}\left\{\mu + (3\mu^2 - 1)a + \left(\frac{15}{2}\mu^3 - \frac{9}{2}\mu\right)a^2 \ldots\right\}. \quad (8)$$

From equations (3), (5) and (8), we have that the amount of reflected light received by the observer is given by

$$\int I \cos\gamma \, d\sigma = \frac{L}{4\pi^2}a^2 \int_{\epsilon-\pi/2}^{\cos^{-1}a} \int_{\sin^{-1}(a \sec\phi')}^{\pi-\sin^{-1}(a \sec\phi')} \left\{\mu + (3\mu^2 - 1)a + \left(\frac{15}{2}\mu^3 - \frac{9}{2}\mu\right)a^2\right\} \cos(\epsilon - \phi') \sin^2\eta \, d\phi' \, d\eta. \quad (9)$$

The limits for η are derived from the consideration of the section of the plane $\phi' =$ constant by the base of the enveloping cone. This is given by $\sin\eta = a \sec\phi'$. The limits for ϕ' indicate that we are considering at first the partial phase, that is, when a part only of the illuminated portion of S_2 is visible to the observer. The integration in the right-hand side of the equation (9) is to be performed with respect to η first.

We substitute for μ from (4) in (9) and integrate (9) term by term, evaluating each partial integral at first in closed form and then expanding the latter in ascending powers of a. Denoting the partial integrals by I_1, I_2 and I_3, we arrive at the following values:

$$I_1 = \int_{\epsilon-\pi/2}^{\cos^{-1}a} \int_{\sin^{-1}(a \sec\phi')}^{\pi-\sin^{-1}(a \sec\phi')} \cos\phi' \cos(\epsilon - \phi') \sin^2\eta \, d\phi' \, d\eta$$

$$= \frac{2}{3}\{(\pi - \epsilon)\cos\epsilon + \sin\epsilon\} - a^2 \sin\epsilon \quad (10)$$

$$I_2 = a\int_{\epsilon-\pi/2}^{\cos^{-1}a} \int_{\sin^{-1}(a \sec\phi')}^{\pi-\sin^{-1}(a \sec\phi')} (3\sin^2\eta\cos^2\phi' - 1)\cos(\epsilon - \phi')\sin^2\eta \, d\phi' \, d\eta = \frac{3\pi}{8}a(1 + 2\cos\epsilon + \cos^2\epsilon) - \frac{\pi}{2}a(1 + \cos\epsilon) + 2a^2\sin\epsilon. \quad (11)$$

$$I_3 = a^2\int_{\epsilon-\pi/2}^{\cos^{-1}a} \int_{\sin^{-1}(a \sec\phi')}^{\pi-\sin^{-1}(a \sec\phi')} \left(\frac{15}{2}\sin^3\eta\cos^3\phi' - \frac{9}{2}\sin\eta\cos\phi'\right) \cos(\epsilon - \phi')\sin^2\eta \, d\phi' \, d\eta = -a^2\sin^3\epsilon. \quad (12)$$

Hence we have for the reflection effect for the partial phase

$$L_p = \frac{L}{4\pi^2}a^2\left[\frac{2}{3}\{(\pi - \epsilon)\cos\epsilon + \sin\epsilon\} + \frac{\pi}{8}a(3\cos^2\epsilon + 2\cos\epsilon - 1) + a^2\sin\epsilon\cos^2\epsilon\right]. \quad (13)$$

We shall now consider the complete phase, that is, when the illuminated portion of S_2 is fully visible to the observer. This will be so long as $\epsilon < \sin^{-1} a$. The limits for ϕ' in this case will be $\cos^{-1} a$ and $-\cos^{-1} a$.

We have for the complete phase

$$I_1 = \frac{2}{3}\pi\cos\epsilon,$$

$$I_2 = \frac{3}{2}\pi a\cos\epsilon - \pi a\cos\epsilon,$$

and

$$I_3 = 3\pi a^2\cos\epsilon - 3\pi a^2\cos\epsilon.$$

Hence we have the reflection effect for the complete phase

$$L_f = \frac{L}{4\pi^2}a^2\left(\frac{2}{3}\pi\cos\epsilon + \frac{\pi}{2}a\cos\epsilon + 0.a^2\right). \quad (14)$$

It should be noted that when $\pi - \sin^{-1} a < \epsilon < \pi$, the star S_2 will be in eclipse phase, that is, no reflected light will be received by the observer.

In a letter to Kopal,[3] Russell mentioned the following useful check which the formulae for the reflection effect should satisfy. The portion of the incident light from S_1 intercepted by S_2 lies within the solid angle

$$\omega = 2\pi(1 - \cos s), \quad (15)$$

where s is the angle which the tangent from the point S_1 to the star S_2 makes with the line S_1S_2 (see Fig. 1).

Now, we have

$$\cos s = \sqrt{1 - a^2} = 1 - \frac{1}{2}a^2 - \frac{1}{8}a^4 \ldots, \quad (16)$$

to our order of approximation.

From equations (15) and (16) we have that the proportion of the incident light of star S_1 intercepted by S_2 is

$$\frac{\omega}{4\pi} = \frac{1}{4}a^2 + \frac{1}{16}a^4 \ldots. \quad (17)$$

As all incident light is reflected back, the proportion of the incident

light received as reflected light by the observer should also satisfy equation (17). We verify below that our formulae satisfy Russell's check.

We evaluate the proportion of reflected light received by the observer in three steps as follows:

(1) Partial phase $(a < \epsilon < \pi - a)$.

$$\frac{2\pi}{L}\int_a^{\pi-a} L_p \sin\epsilon\, d\epsilon = \frac{a^2}{2\pi}\int_a^{\pi-a}\left[\frac{2}{3}\{(\pi-\epsilon)\cos\epsilon + \sin\epsilon\} + \frac{\pi}{8}a(3\cos^2\epsilon + 2\cos\epsilon - 1) + a^2\sin\epsilon\cos^2\epsilon\right]\sin\epsilon\, d\epsilon$$

$$= \frac{1}{4}a^2 - \frac{5}{48}a^4.$$

(2) Complete phase $(0 < \epsilon < a)$.

$$\frac{2\pi}{L}\int_0^a L_f \sin\epsilon\, d\epsilon = \frac{a^2}{2\pi}\int_0^a \left(\frac{2}{3}\pi + \frac{\pi}{2}a\right)\cos\epsilon\sin\epsilon\, d\epsilon$$

$$= \frac{1}{6}a^4.$$

(3) Eclipse phase $(\pi - a < \epsilon < \pi)$.

There is obviously no contribution to the reflected light in this phase.

Hence the proportion of the incident light received as reflected light by the observer is

$$\frac{1}{4}a^2 + \frac{1}{16}a^4 \ldots . \qquad (17)$$

Our formulae for the reflection effect therefore satisfy Russell's check to the adopted order of approximation.

The author desires to offer his sincere thanks to Dr. Zdeněk Kopal for suggesting the problem and for considerable helpful criticism during the course of the work. The author's best thanks are also due to Professor Henry Norris Russell for illuminating discussion, to Professor Harlow Shapley for the very valuable privilege of working at the Harvard College Observatory, and to Mr. Arthur Hoag for kindly drawing the figures.

Summary.—The reflection effect for a point illuminating source has been evaluated, correct to the fourth power of the ratio of the radius of the illuminating star to the distance between the two stars, on the hypothesis of a cosine law of reflection.

[1] *M. N.*, **87**, 43 (1926).

[2] *An Introduction to the Study of Eclipsing Variables*, 1946, p. 155.

[3] Dr. Kopal in his *Eclipsing Variables* (*loc. cit.*, p. 155) has given an outline of a solution initiated by Takeda (*Kyoto Mem.*, **A17**, 197 (1934)). But, as Prof. Russell pointed

out in a letter to Dr. Kopal, the upper limit of $\pi/2$ in Dr. Kopal's integral of the reflection effect extends it beyond the convergent cone. At Dr. Kopal's suggestion, the author has rigorously evaluated, in this paper, the integral with the proper limits.

[4] *Loc. cit.*, p. 154.

[5] Kopal, *Annals, New York Academy Sciences*, **41**, 25 (footnote), 1941.

[6] Kopal, *Eclipsing Variables*, p. 164.

THE STIMULATORY ACTION OF CERTAIN FRACTIONS FROM BACTERIA AND YEAST ON THE FORMATION OF A BACTERIAL VIRUS

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Recent experiments have shown that the formation of bacterial viruses and the response of the host cells depend upon the composition of the culture medium. Wahl[1] has shown that some coli bacteriophages may be formed in synthetic medium without lysis of the host provided a certain concentration of thiamin and calcium is added to the medium. Raising the concentration of thiamin results in lysis of the host although no more virus is formed than without lysis. Experiments from this laboratory[2] have shown that *Staphylococcus muscae* phage can also be released in synthetic medium without lysis of the host. The addition of a substance present in veal infusion results in lysis of the host in the latter system. Fowler and Cohen,[3] using a coli phage, have been able to increase the virus yield per cell by varying the composition of the medium. Maurer and Woolley[4] have reported that the addition of apple pectin to synthetic medium permits the release of *E. coli* phage without cellular lysis.

It has now been found that a fraction from yeast or from the bacterial cell stimulates the formation of a bacterial virus. This fraction has a high concentration of ribonucleoprotein and has been referred to as the ribonucleoprotein fraction by other workers.[5] The phage-stimulating property parallels the ribonucleoprotein fraction during purification. Splitting the ribonucleoprotein into free nucleic acid and protein causes complete loss of the phage-promoting activity. In view of these results, our working hypothesis is that the active substance is a ribonucleoprotein until experiments may prove otherwise.

A ribonucleoprotein fraction from yeast has recently been shown to stimulate the formation of adaptive enzymes in yeast.[6] This nucleoprotein fraction has also been found to accelerate the formation of a bacterial

virus. These results, together with a comparison of adaptive enzyme formation and bacterial virus formation, will be described in this paper.

Experimental.—The *Staphyiococcus muscae* phage system described previously was used in all the experiments.[7] The cells and virus were grown as reported earlier.[2] Bacteria and phage were determined as in earlier experiments.[7] The synthetic medium was the same as that described previously.[2] One-step growth curves were carried out according to Delbrück and Luria.[8]

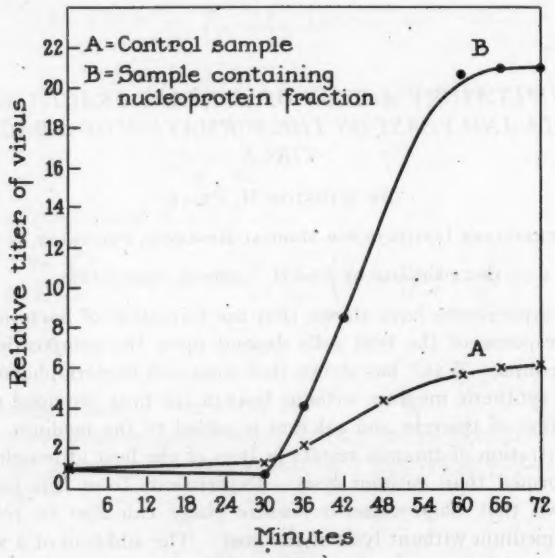

FIGURE 1

The effect of the bacterial ribonucleoprotein fraction on the formation of bacterial viruses. The bacterial cells were washed off a 20-hour old veal infusion agar slant and prepared as described under Methods. Two tubes of synthetic medium containing 2.0 mg. of hydrolyzed casein were inoculated with 5.8×10^7 cells per ml. Tube A was the control and tube B received 0.1 ml. of the ribonucleoprotein fraction containing 0.7 γ of nitrogen per ml. After 1 hour of incubation both tubes contained 6.2×10^7 cells per ml. To each tube 0.1 ml. of a virus solution was then added to give a final titer of 4.8×10^7 particles per ml. The tubes were shaken 18 minutes and then diluted 1:2000 in their respective media. The control tube showed 43% of the virus adsorbed and the tube containing the nucleoprotein showed 40.7% of the virus adsorbed. Samples were taken from the diluted tubes for virus assay at various times.

Most of the nucleoprotein fractions were prepared from bacterial cells first ground with powdered glass and then extracted with water. After centrifugation the supernatant fluid was adjusted to pH 4.0 with 10%

acetic acid and 1 volume of alcohol added. The precipitate was collected, resuspended in water and adjusted to pH 6.3 with 0.5 *M* $NaHCO_3$. This solution was adjusted to pH 3.7 with 10% acetic acid. The precipitate was collected and the acid precipitation repeated. The yeast ribonucleoprotein fraction was prepared from fresh baker's yeast in a similar manner, except that the crude water extract was dialyzed against water for 10 hours before the alcohol fractionation. All operations were carried out at 5°.

Results.—The addition of the nucleoprotein fraction does not increase the per cent adsorption of the virus to the cell nor does it decrease the minimum latent period (Experimental Procedure in figure 1). It does increase the yield of virus per cell. As shown in figure 1, for the first 30 minutes the phage count, which represents the unadsorbed phage and the

TABLE 1

THE EFFECT OF VARYING CONCENTRATIONS OF THE BACTERIAL NUCLEOPROTEIN FRACTION ON THE FORMATION OF THE BACTERIAL VIRUS

The same conditions were used as in figure 1. The values below represent the maximum titer at the end of a one-step growth curve. The initial titer was 2.1×10^4 plaque counts per ml.

SAMPLE	γ *N* OF RIBONUCLEOPROTEIN FRACTION ADDED PER ML.	% ADSORPTION OF VIRUS TO CELL	FINAL PLAQUE COUNTS PER ML.
1	..	40.2	1.1×10^5
2	0.22	42.1	2.1×10^5
3	0.44	38.6	3.1×10^5
4	0.88	41.3	5.3×10^5
5	1.6	39.1	5.6×10^5

adsorbed phage, remains constant. This period is called the minimum latent period.[8] At the end of 30 minutes, the phage titer goes up for 30 minutes as the virus particles are released by the infected cells. Due to the high dilution step used in the one-step growth curve, the phage titer no longer increases after the initially infected bacteria have released their phage, since there is no readsorption of the released virus particles to new cells. The average yield of viruses per cell may be calculated by the equation[8] below.

$$\text{Average burst size} = \frac{\text{final virus} - \text{initially unadsorbed virus}}{\text{initial virus} - \text{initially unadsorbed virus}}.$$

In the experiment shown in figure 1 the control tube formed an average of 7 viruses per cell and the tube containing the nucleoprotein fraction, 31 virus particles per cell. In the hundreds of tests that have been run, the nucleoprotein fraction from yeast or bacteria has increased the relative phage titer from 2 to 30 times over the control. The average stimulation

is generally fourfold. It should be noticed that these tests are run with cells in the resting phase. Cells in the log phase form more phage than those in the resting phase and under these conditions the nucleoprotein fraction has only a slight effect, increasing the formation of phage not quite two times. It appears that this substance may be synthesized by rapidly growing cells. The experiment in table 1 shows the effect of varying concentrations of the nucleoprotein fraction on the formation of the virus.

The ultra-violet absorption curve of a highly purified fraction of the yeast ribonucleoprotein fraction shows a sharp maximum at 2600 A. U. and a minimum at 2450 A. The minimum at 2450 A is characteristic of nucleoproteins rather than free nucleic acid. The minimum of free nucleic acid is at 2300 A.[9] Splitting the nucleoprotein into free nucleic acid and protein causes a complete loss of phage-promoting activity. In the present state of purity, the phage-stimulating activity of the ribonucleoprotein fraction is not destroyed on incubation with trypsin, chymotrypsin, pepsin, ribonuclease or desoxynuclease. It is, of course, not dialyzable. It is not precipitated by cold trichloroacetic or metaphosphoric acid, is very poorly precipitated by ammonium sulfate, and is maximally precipitated by acid at around pH 3.7. It is not spun down by centrifuging 1 hour at 24,000 r. p. m. at pH 6.8 in a Bauer-Pickels air-driven centrifuge. The best bacterial preparation contained 15.3% nitrogen and 2.6% phosphorus. The nucleic acid is all of the ribose type.

Ribonucleoprotein fractions prepared from calf thymus, pancreas and liver are inactive. The virus, which contains mostly if not all desoxynucleic acid, inactivated by mild heating or acid, has no phage-promoting activity under our conditions. The conditions used to inactivate the phage do not inactivate the ribonucleoprotein factor. Tobacco mosaic virus, which is a ribonucleoprotein, shows no phage stimulating activity in our system.

Several months ago in a short abstract Reiner and Spiegelman announced the isolation from yeast of a ribonucleoprotein fraction which stimulated the formation of adaptive enzymes in yeast.[6] In view of the great similarity between their substance and the substance being studied in this laboratory, their adaptive enzyme fraction was tried in our phage system. Table 2 shows that it acted exactly like the fraction isolated by us and had approximately the same activity per mg. of nitrogen as our fraction. All three purified preparations sent to us by Dr. J. Reiner were active in our system. Our compound is not active in the adaptive enzyme system. This is not surprising since their substance must be isolated in the presence of galactose.

Discussion.—The experiments reported in this paper show that the addition of a fraction, from the host bacterium or from yeast, containing ribonucleoprotein increases the virus yield per cell of infected bacteria.

The ribonucleoprotein fraction isolated from yeast by Reiner and Spiegelman[6] which stimulates adaptive enzyme formation is very similar to the substance isolated in this laboratory. Indeed, it may replace it in stimulating phage formation.

It is difficult at the present time to understand the mechanism involved in the stimulation of the formation of a phage containing desoxynucleic acid by the ribonucleoprotein fraction. A theory has been proposed by Spiegelman[10] to account for the action of his ribonucleoprotein fraction in enzyme formation. We have no evidence for or against his theory at the present time.

It appears to us that two possibilities exist: (1) the factor acts as a precursor-like or nutrient substance or (2) it effects some reaction necessary in virus synthesis. As a working hypothesis, the latter theory is being tested. From the work of Caspersson[11] and Brachet[12] it appears that

TABLE 2

THE EFFECT OF THE YEAST ADAPTIVE ENZYME FACTOR ON THE FORMATION OF THE BACTERIAL VIRUS

The same conditions were used as described in figure 1. The values below represent the maximum virus titer at the end of a one-step growth curve. The amounts of nitrogen used represent that concentration which gave 50% stimulation. The initial titer was 3.1×10^4 plaque counts per ml. The adsorption was approximately 40% in all samples.

SAMPLE	ADDITIONS	FINAL PLAQUE COUNTS PER ML.
1	.....	1.1×10^8
2	Ribonucleoprotein fraction isolated in this laboratory (1.1γ N per ml.)	2.7×10^8
3	Enzyme stimulatory factor (0.81γ N per ml.)	3.1×10^8

ribonucleic acid is somehow concerned in protein formation. This, together with the observation that the ribonucleoprotein fraction also increases enzyme formation, makes it a likely hypothesis that the fraction is somehow accelerating protein synthesis. Chemical determinations of cells grown in the presence of this fraction have so far shown no differences over control cells, and cells, even in the lag phase, do not multiply faster in the presence of the compound.

Another interesting point brought out by these experiments is the similarity between adaptive enzyme formation and bacterial virus formation. This similarity was pointed out by Northrop[13] and, although this view was not very popular, it should be reëxamined in the light of new experimental evidence. An adaptive enzyme is formed by cells when grown in the presence of its substrate just as cells form phage when grown in the presence of phage. Before the production of either adaptive

enzyme[14] or virus,[8] there is a lag period. Both enzyme[14] and virus[15] may be formed in non-viable cells. Cells which have the ability to multiply rapidly appear to form adaptive enzymes[16] and viruses[17] better than old cells. A ribonucleoprotein fraction from yeast stimulates both virus and adaptive enzyme formation. One of the great differences that has existed between adaptive enzymes and viruses has been the destructive effect of the virus on the host. Even this difference, however, is not a very sharp one, since it has been found that the pathological response of the host to the virus may be modified by varying the medium.[1, 2, 4]

Competitive interactions in the cell exist in the formation of both adaptive enzymes and bacterial viruses. Thus Spiegelman[18] has shown that the formation of an adaptive enzyme in yeast may cause decreases in the other enzyme systems of the cell. In the formation of bacterial viruses in the *E. coli* system, Cohen[19] has presented evidence that the formation of the virus prevents the synthesis of cellular constituents. The very interesting observations of Monod[20] on the formation of adaptive enzymes in bacteria are important in this connection. On exposing a cell to two sugars simultaneously, only one adaptive enzyme was synthesized. A similar situation in phage formation has been found by Delbrück and Luria[8] who infected a bacterium with two different viruses and found that only one multiplied. Thus in both adaptive enzyme formation and bacterial virus multiplication there may exist a mutual exclusion effect. Finally, there are the observations, with a few exceptions, that in bacteria, viruses[19] and adaptive enzymes[20] both need a source of external nitrogen for their formation.

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*VITAMIN K_5 AS AN INHIBITOR OF THE GROWTH OF FUNGI AND OF FERMENTATION BY YEAST**

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The present report is concerned with new biological observations on one of the vitamin K analogues, vitamin K_5, known chemically as 2-methyl-4-amino-1-naphthol hydrochloride. This compound which has been used with remarkable success as a water-soluble form of antihemorrhagic vitamin,[1] is characterized by low toxicity for animals, the LD_{50} for mice being approximately 750 mg./Kg. when administered orally[2] or intraperitoneally.[3] Discussion of the chemistry and pharmacology of vitamin K and related compounds and methods of laboratory synthesis of different analogues of the former have been published.[4, 5, 6] Due to their inhibitory action on the formation of acid in saliva, both vitamins K_3 (2-methyl-1,4-naphthoquinone) and K_5 may find practical application in prevention of dental caries.[7, 8] Vitamin K_3 and 2-methyl-1,4-naphthohydroquinone retard the growth of *Penicillium notatum*,[9] of other fungi, and of yeasts.[10] Vitamin K_5, however, appears not to have been studied for antifungal properties.

The vitamin K_5 (2-methyl-4-amino-1-naphthol hydrochloride) used in the following experiments was synthesized as previously described by one of us[11] with slight modifications to improve the yield.

Inhibition of Growth of Fungi.—Penicillium notatum and *Trichophyton mentagrophytes* were selected for study as representatives of non-pathogenic and pathogenic fungi, respectively. A heavy inoculum of spores of *P. notatum* was seeded into each of several mushroom spawn bottles containing a corn steep liquor and mineral salt culture solution to which different amounts of vitamin K_5 were added. The initial pH of all solutions was adjusted to 5,5. Each concentration was studied in duplicate or triplicate in each experiment. The experiment was repeated three times, always with the same result. Growth of the mold was inversely

related to the concentration of K_5: a concentration of 100 mg./L. (100 p. p. m.) suppressed all growth. This is shown in figure 1 which is a reproduction of a photograph taken two weeks after inoculation.

The effect of K_5 on *T. mentagrophytes* was studied using Sabouraud's medium and the well-known agar cup-plate technique. The Petri dishes used were 9 cm. in diameter: the central wells cut in the agar with a sterile

FIGURE 1

Growth of *Penicillium notatum* on a culture solution containing corn steep liquor, mineral salts, and different amounts of vitamin K_5. The concentrations of K_5 are indicated in mg./L. at the bottom of each bottle. Photograph taken two weeks after inoculation. Growth was markedly retarded by a concentration of 50 mg./L. and was checked completely by a concentration of 100 mg./L.

cork borer were 1.1 cm. in diameter. A 1 per cent solution of K_5 checked growth of the fungus on the entire surface of the plates when placed in the central wells. A 0.1 per cent solution (1000 p. p. m.) produced zones that averaged 6.2–6.3 cm. in diameter. It seems probable that in broth cultures where the factor of diffusion of the comparatively large molecule through agar would be eliminated, lower concentrations might suffice

to prevent growth of this organism, since the compound completely checked growth of the fungus when incorporated in the nutrient agar in a concentration of 0.001 per cent.

Similarly vitamin K_5 was observed to be endowed with strong fungistatic

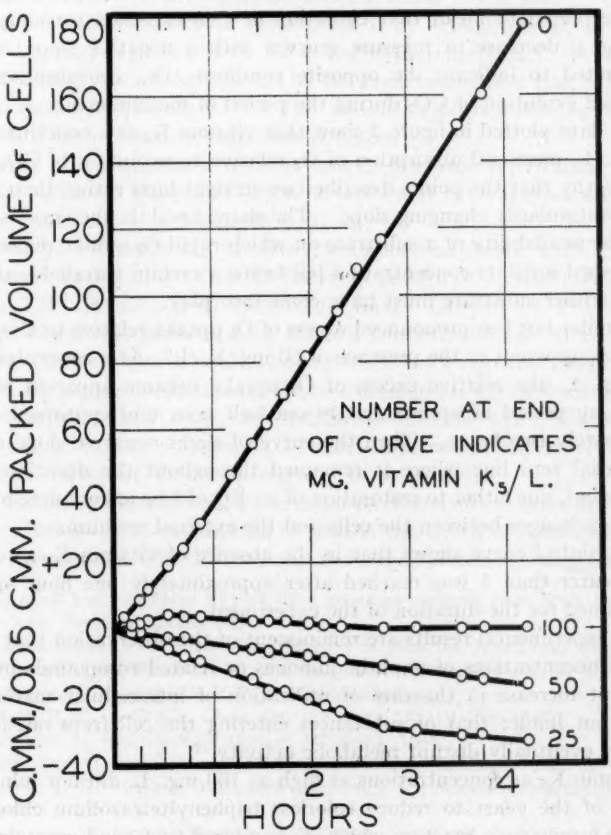

FIGURE 2

Change in gas pressure produced by yeast cultures in 10 per cent glucose with different concentrations of vitamin K_5.

activity when *Microsporum canis*, *M. audouini* and the plant pathogen *Botrytis allii* were employed as test organisms.

Inhibition of Yeast.—The effect of different concentrations of vitamin K_5 on production of CO_2 by yeast (*Saccharomyces cerevisiae*) in 10 per cent

glucose was studied manometrically by means of the Warburg technique. All solutions were adjusted to pH 4.8. The averaged results of several experiments are shown in figure 2. Since the suspensions of yeast cells were placed in vessels with no KOH or other agent to absorb the gas that was liberated, an increase in pressure (curves with a positive slope) may be interpreted to mean that evolution of CO_2 exceeded consumption of O_2, and a decrease in pressure (curves with a negative slope) may be interpreted to indicate the opposite condition, i.e., consumption of O_2 exceeded evolution of CO_2 during the period of measurement.

The data plotted in figure 2 show that vitamin K_5 at a concentration of 25 mg./L. increased absorption of O_2 relative to evolution of CO_2. It is noteworthy that the points describe two straight lines rather than a curve with continuously changing slope. The sharp break in the curve suggests that the availability of a substrate on which rapid O_2 uptake depends was diminished until its concentration fell below a certain threshold, at which time another substrate must have come into play.

A similar but less pronounced excess of O_2 uptake relative to CO_2 evolution was apparent in the presence of 50 mg. K_5/L. At a concentration of 100 mg./L. the relative excess of O_2 uptake became apparent after an initial lag period of approximately one-half hour and continued for approximately two hours. Then the curve of slight negative slope reached the initial zero line where it remained throughout the duration of the experiment, due either to restoration of an RQ of 1 or to complete blocking of gas exchanges between the cells and the external medium.

The control curve shows that in the absence of vitamin K_5 a constant RQ greater than 1 was reached after approximately one hour and was maintained for the duration of the experiment.

The experimental results are reminiscent of the observation that appropriate concentrations of naphthoquinones or related compounds induce a transient increase in the rate of utilization of intracellular materials in algae, but hinder that of substances entering the cell from outside and thereby eventually disrupt metabolic activity.[12]

Vitamin K_5 at concentrations as high as 100 mg./L. did not inhibit the ability of the yeast to reduce colorless triphenyltetrazolium chloride to the red formazan, a reaction which is considered to depend upon dehydrogenase systems requiring coenzymes I and II.[13] Conversely, solutions containing 25 mg. or more of K_5 per L. inhibited the indophenol oxidase system in yeast. Normally yeast cells promote rapid formation of indophenol-blue from a mixture of dimethyl-*p*-phenylene-diamine and thymol, but after having been exposed to solutions containing 25 to 100 mg. K_5/L., they not only tend to prevent formation of indophenol-blue but, if brought into the presence of the dye, they reduce it.

Discussion.—Our experimental results with vitamin K_5 are in accord

with those of Ball, *et al.*,[14] who found that substituted naphthoquinones prevent the oxidation of *p*-phenylenediamine and interfere with the respiratory systems at a redox potential between those at which cytochrome *C* and cytochrome *B* operate. More specifically vitamin K_5 may be assumed to interfere with the component which Bach, *et al.*,[15] consider necessary for the reduction of cytochrome *C* by cytochrome *B*, and which Slater[16] tentatively identified as a haematin compound. It may be suggested that vitamin K_5 may act by the same mechanism that Schönberg and co-workers[17] postulated for the various naphthoquinones, viz., by virtue of a high oxidation-reduction potential.

Because of its fungistatic activity and its low toxicity, vitamin K_5 may prove of value for the preservation of foods and beverages as well as for clinical treatment of dermatophytic fungi.

Summary.—Vitamin K_5 (2-methyl-4-amino-1-naphthol hydrochloride) at a concentration of 0.1 Gm./L. prevents growth of *Penicillium notatum* NRRL 1249.B4 on a rich culture medium. Growth of *Trichophyton mentagrophytes* was prevented by K_5 at a concentration of 10 Gm./L. in agar cup-plate tests or by 0.01 Gm./L. when the compound was incorporated in the nutrient substratum. The compound is strongly antagonistic also to *Microsporum canis*, *M. audouini*, and *Botrytis allii*. Concentrations of 25 mg. or more of K_5/L. checked fermentation by yeast (*Saccharomyces cerevisiae*) in a 10 per cent glucose solution.

The results are interpreted in terms of altered redox potentials.

The possible use of vitamin K_5 in clinical treatment of dermatomycoses and as a preservative in the food and beverage industries is suggested.

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INACTIVATION OF ENZYME-SUBSTRATE FILMS BY SMALL DOSES OF X-RAYS

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The analysis of the biological effects of radiations in terms of their effects on simpler biochemical systems is rendered difficult by the fact that most biochemical systems seem so insensitive to radiations. In particular, chromosomes are often affected, structurally and genetically, by doses smaller by orders of magnitude than those which affect enzyme solutions, virus preparations, etc., measurably. This apparent paradox contains certain clues for the further analysis of biological effects. It focuses attention on the chromosomes as the sensitive agents in the cell. This apparent sensitivity is based in part on method. Chromosomal effects are often expressed as visible changes, so that a single effect may be detected. The genetic units in the chromosome are unique, so that the consequences of a single event at the molecular level can be far-reaching and persistent. This contrasts with the statistical nature of most chemical measurements.

But the question of the radiation sensitivity of chromosomes involves factors not represented in an enzyme or virus solution, most clearly evident where a single radiation event produces a break in a microscopic structure. The chromosome is a continuous "solid" body, varying in both structure and sensitivity through its own cycle and according to experimental conditions. In attempting to visualize the effects of radiation, the structural factor has to be introduced. Therefore it would be desirable to set up a biochemical system in which the factor of intermolecular structure was present and in which the variables encountered by the chromosomes could be tested. If such a system showed a high radiation sensitivity, this would in itself suggest a relation between structure and sensitivity.

We have, in accordance with these requirements, investigated the radiation sensitivity of surface films containing a proteolytic enzyme and its substrate. Mazia, Hayashi and Yudowitch[1] have described the structure and enzymatic activity of such a system involving pepsin and albumin, and have considered possible parallels with chromosome structure and activity. Preliminary experiments showed that this system was indeed sensitive to radiations. X-ray doses of the 50–150 r order produced extensive inactivation.

The sensitivity seemed to depend on the structural configuration, for the same doses applied to the solutions from which the films were prepared had no detectable effect. The sensitivity might depend on alterations of the molecules on spreading, on the participation of sensitive intermolecular bonds, not present in the solution, in the reaction, or merely on the geometry of the exposure of the films to the irradiated medium. All of these possibilities may also be implicated in the sensitivity of chromosomes, and all of them would be absent in experiments on solutions. The present series of investigations is an exploration of these structural factors in radiation sensitivity. The possible analogies between the actual structure of the films and of chromosomes are not essential to such an exploration.

Methods.—Each experiment consists of the following steps. (1) A film containing both enzyme and substrate is prepared by spreading at a pH at which the activity of the enzyme is negligible. (2) The film is irradiated. (3) The film is compressed into a fiber which is removed and washed. (4) The enzymatic activity in the fiber is estimated by bringing the fiber to the pH optimum of the enzyme and timing its self-digestion as observed microscopically.

Solutions.—The starting solutions consisted of mixtures of crystalline egg albumin, prepared by the method of LaRosa,[2] and crystalline pepsin (Lehn and Fink Co.) in water. The albumin was dialyzed in the cold and frozen dried. As this treatment produced pepsin preparations of varying activity, we used the pepsin-$MgSO_4$ mixture without further purification, estimating its protein content by the method of Robinson and Hogden.[3] The total protein concentration used was 0.2 per cent. A small amount of isopropyl alcohol was used to improve spreading.

Preparation of Films.—The films were spread over 10-fold diluted MacIlvaine buffer, the final pH being 4.0, in a Cenco Hydrophil tray. Pyrex-redistilled water was used. The protein solution was measured onto the previously swept surface with a 0.1 ml. pipette. In most of the experiments, 0.05 ml. of the solution, a slight excess over what would be required to cover the surface under ideal conditions, was used. This meant that there was a small amount of surplus protein, about 0.03 mg. of albumin and about 0.001 mg. of pepsin, dissolved in the liter or so of buffer under the film. The buffer was changed for each experiment, so that there would be no accumulation of dissolved protein. The possibility of misinterpretation of the results because of the presence of dissolved enzyme will be considered later.

Irradiation.—The film trough was placed under the x-ray tube so that the target was directly over the center of the film. The standard area irradiated was 18 × 32 cm. The dose rate was controlled by varying the target-to-film distance, but, to insure covering the film, this was never less than 20 cm.

The Coolidge tube was operated at 150 kv. and 4 amperes with a 1-mm.

Al filter. Dose was not measured for each experiment, but was calculated from calibrations made with a Victoreen dosimeter set at various points in the film tray, the tray being filled with paraffin. By this means, allowance could be made for back-radiation, and for variations inherent in the geometry of the system. Doses given in the data below are those immediately below the target, and therefore represent the maximum received by the films.

Test of Activity.—The activity of pepsin-albumin films may be detected and measured microscopically. The film is compressed into a fiber by bringing the barriers together, and the fiber is removed and washed as free as possible of solutes from the trough with distilled water. The washed fiber is then placed in HCl at pH 1.5 and observed microscopically. Fibers containing active pepsin are observed to digest themselves and disappear rapidly. Control fibers containing albumin and pepsin in a 40 to 1 ratio digest in 30 to 50 seconds at pH 1.5 and 25°C. Mazia and Hayashi[4] have tested this microscopic method by comparison with measurements on the rate of liberation, under similar conditions, of trichloracetic-soluble split products, following the methods of Anson.[5] The results have invariably been parallel; the slower the disappearance of the fibers, the lower is the rate of release of split products. While the time required for visible digestion may not be a precise measure of the *rate* of digestion, we have never encountered a case where visible digestion was not accompanied by release of split products or where a fiber that did not visibly digest gave a full yield of split products. We consider, therefore, that a slowing down or complete inhibition of visible digestion is a measure of inactivation, and this was the measure used in testing the effects of irradiation. For the present, our chemical method is not suitable for the type of experiment described in this paper, since it requires about 1 mg. of dried fiber.

Results.—Rate of Digestion as a function of x-ray Dose.—The effects of radiation on digestion-time are shown in figure 1. The two curves represent films prepared from solutions containing pepsin-albumin ratios of 20:1 and 40:1. Each point represents a completely independent experiment. We shall not at this time consider the source of variability in the observations, except to mention that later work indicates that sensitivity is very dependent on surface pressure which was not controlled in the experiments shown in figure 1. The trend of the results is clear enough. Effects of doses of the order of 50 r may be detected and doses over 150 r produce the maximum effect that can be observed: complete failure of the compressed film to digest. The results suggest very strongly a "threshold" effect. In terms of delay in digestion time, doses below 50 r are not very effective compared with the same increments of dose above 50 r.

Measurement of Effect.—While the results given do show that we are dealing with a highly radiosensitive enzyme system, the bare data do not

permit a quantitative evaluation of the effect. For this purpose it would be desirable to express the results as the proportion or number of active units

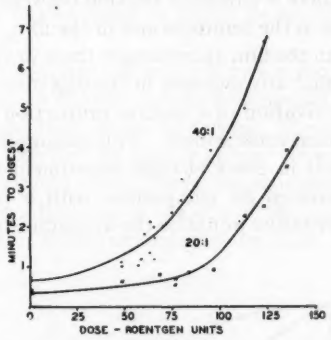

FIGURE 1

Relation between digestion rate and x-ray dose. Each point represents an independent experiment. Ratios are albumin-pepsin ratios in solutions from which films were prepared.

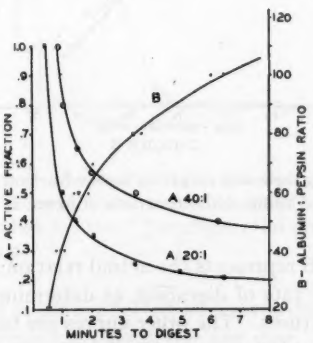

FIGURE 2

Relation between enzyme content of films and digestion rate. Curve *B* represents digestion rates for various albumin pepsin ratios (Scale *B*). Ratios are those in solutions from which films were prepared. Curves *A* (read against scale *A*) are calculated from Curve *B*. Enzyme content of 20:1 and 40:1 films are taken as unity, digestion rate is plotted against fractions of initial enzyme content.

affected. This is possible if a relation can be found between the number of active units and the digestion rate. Such a "calibration" is obviously possible, for all that is necessary is to determine the digestion rate of films pre-

pared from solutions containing various substrate enzyme ratios. The results of such a calibration are given in figure 2.

The use of this calibration depends on certain assumptions. First, it has to be assumed that there is a definite relation between the enzyme and substrate concentrations in the solutions and in the film. It is assumed, in the plot, that the ratios in the film approximate those in the solutions. Second, it must be assumed that any decrease in the digestion rate after irradiation results from the inactivation of a certain proportion of enzyme molecules rather than other conceivable causes. This assumption is difficult to test. It is, however, present in every enzyme experiment where the amount of active enzyme is deduced by comparison with a concentration-activity curve. The only alternative would be the impracticable one of isolating the active fraction.

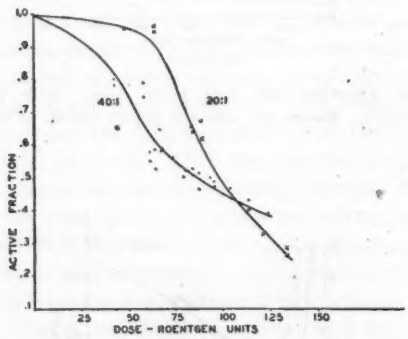

FIGURE 3

Relation between x-ray dose and surviving active fraction of enzyme in film. Data from figure 1 are replotted using calibration data of figure 2.

In figure 2, Curve *B* represents the actual relation between the substrate enzyme ratio and the rate of digestion, as determined by separate experiments on various mixtures. The other curves are based on the same data and are introduced merely for convenience. They represent the relation between the active fraction of the enzyme and digestion rate where certain substrate-enzyme ratios are taken as unity.

These curves may then be used to express the radiation data of figure 1 in terms of the surviving fraction of the initial activity. Obviously, they can also be used to calculate the number of units inactivated, using the area of the film and the molecular weights of the proteins.

Figure 3 shows the radiation data plotted as dose against active fraction. If the calibration of the method is valid, this plot establishes the high sensitivity of the system in an absolute sense, for it is seen that a dose of 100 r

destroys more than half of the activity present. Certain other points are suggested by the plot.

1. The "threshold" effect suggested by figure 1 remains. The point of deflection is difficult to determine as might be expected where individual films vary in sensitivity.

2. There is an unexplained difference between the two films of different composition. The threshold effect is less pronounced in the film with a 40:1 substrate enzyme ratio than in the 20:1 film. Moreover the whole dose-effect relationship is different for the two films. If we were dealing with a "hit" relationship, we could expect the curves to coincide, since the fraction affected would not be a function of the size of the population. If we were dealing with an indirect action as described by Dale, it would be expected

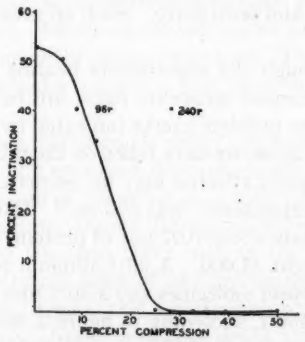

FIGURE 4

Relation between surface compression and radiation sensitivity. Compression given in units of area, not pressure. Inactivation calculated from calibration in figure 2. Curve represents inactivation by 96 r. The single point gives inactivation by 240 r at 28 per cent compression.

that the total number of units affected by a given dose would be independent of the number present. This is not the case. It is obvious that the composition affects the sensitivity in a way that is not simple.

Effect of Compression on Sensitivity.—These films are more sensitive to radiation than would be predicted from their composition alone, and the structural factors in their sensitivity have to be taken into account. One possibility is that the spreading of the enzyme molecule (however incomplete) may increase its sensitivity. Another is that the structural continuity may be a factor. These variables may be tested by determining the effect of surface compression on radiation sensitivity. In these experiments, the films were spread as already described, compressed to a given area, irradiated, then fully compressed and tested for digestion rate. The results are

given in figure 4. There is evidently a sharp, discontinuous effect. Sensitivity begins to fall off at about 10 per cent compression, and at about 28 per cent compression is lowered to the point where the effects of a dose of 96 r are not detectable. Sensitivity is only lowered, not destroyed. At 28 per cent compression, 240 r produce about the same effect as 96 r at 13 per cent compression. If area of exposure to irradiated medium determined the sensitivity, one would expect some simple proportionality between area and radiation effect instead of the discontinuous relationship observed. It seems clear that, regardless of the mechanism of the radiation effect, the effect is on the film itself rather than on dissolved enzyme and that the sensitivity is determined by the physical configuration of the film. Obviously, the "compression" shown in the data is only a very rough index of changes in surface pressure, and it is necessary to investigate the relationship between actual pressure and sensitivity. Such an investigation is now under way.

Calculations.—Although the experiments bearing on the mechanism of x-ray action on the enzyme substrate films will be reported in a second paper, the nature of the problem can be indicated by some preliminary calculations. In making these, we have relied on the tables computed by Lea.[6] First, the number of units affected may be estimated from figure 3. The film area used in our experiments was 576 cm.^{2} Film pressure determinations show this to contain about 0.07 mg. of protein, or a total of 10^{15} molecules of molecular weight 34,000. A 20:1 albumin pepsin film would contain about 5×10^{13} pepsin molecules and a 40:1 film half this number. In orders of magnitude, 100 r, according to figure 3, would produce inactivation equivalent to the removal of 10^{13} pepsin molecules.

The expected number of ionizations in the film itself may be estimated on the basis of the mass of protein, neglecting geometric considerations which would make the true figure lower than the estimate. For the film containing 0.07 mg. of protein, 100 r of 0.15 Å x-rays would yield about 10^{10} ionizations, a figure lower by orders of magnitude than the estimated number of pepsin molecules inactivated.

If the effect is through ionization products in the medium, the calculation has to be made on the basis of unstable products of water decomposition. Experiments in which films were spread over solution that had previously received large doses of radiation showed no differences from controls, and therefore the formation of adequate concentrations of stable products must be excluded. Using Lea's data again, it is calculated that the number of radicals produced by 100 r is of the order of 10^{14} per cc. One may calculate the time that would be required for 10^{13} radicals to reach the film if they were stable. The equation for diffusion across a plane from a semi-infinite column into a column of zero concentration is given by Jacobs[7] (equation 104). We use as diffusion constant the value of 2×10^{-5} cm.^{2} sec.$^{-1}$, the

constant for D_2O in H_2O. The calculation shows that it requires $10^{-4}-10^{-3}$ seconds for 10^{13} radicals to reach the surface. If, then, radicals produced by the decomposition of water were the effective agents, the simple calculation would require their half-life to be of this order. If only radicals finding a pepsin molecule were effective, the time would be 20–40 times greater. Lea estimates 10^{-7} seconds as their half-life in water, and less in the presence of solutes such as citric acid which was present in 0.01 *M* concentration in our experiments.

On the face of these considerations, our results would require the assumption that the effects of a single radiation event, whether in direct action through ionization or indirect action through ionization products, are spread over a considerable number of pepsin molecules.[8] These calculations alone, however, cannot be considered to be a demonstration of the spread of the effect. Lea's estimate of the half-life of radicals produced by irradiation of water may be too low. Moreover, it may not be valid to base the number of inactivations on numbers of pepsin molecules. The film may in fact be a complex polymer, where the scope of a unit inactivation would be difficult to define.

Discussion.—We have restricted ourselves to the description of the radiation sensitivity of the film enzyme-substrate system, and will present data bearing on the mechanism of the effect in a subsequent publication. The basic fact is that the pepsin-albumin films described are unexpectedly sensitive to x-rays, doses of less than 200 r causing the maximum measurable inactivation and doses of the order of 50 r producing readily detectable inactivation. In absolute terms, this sensitivity exceeds that of biological systems, where we do not obtain, with such doses, such large percentages of all possible effects.

The dose-effect curves, with the indicated "threshold" effect, suggest nothing so much as "multiple hit" curves, but their analysis must await the decision as to the type of mechanism causing the effect.

The interest of these observations rests not so much on the sensitivity itself, which might conceivably be duplicated in dilute pure solutions, as on the fact that the sensitivity is obtained in a situation conceptually comparable to that in chromosomes. By organizing the enzyme system into a solid film we are liberated from the restrictions of a dilute solution. The films are as "concentrated" as can be imagined and may contain any number of components. The only possible "protective" action that need be considered is that of the solutes in the adjacent liquid phase. Without pressing detailed analogies between films and cellular structures, it is obvious that with the films certain variables arising in cytological problems may be investigated at a biochemical level. Examples are the question of the spreading effect of a single radiation event within a structure, the range of ionization products, and, particularly, the relation between physical structure and

radiation sensitivity. The aim of further work along these lines is to find ways of picturing biological effects of radiation and premises to be used in their interpretation.

Summary.—(1) Pepsin-albumin films may be inactivated by small doses of x-rays. Doses of about 100 r produce about 50 per cent inactivation.

(2) The sensitivity to radiation depends on the physical configuration of the molecules. It may be varied by surface compression.

(3) Calculations suggest that the effects of a single radiation event (ionization or radical production) may be spread to include a large number of enzyme molecules.

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NOTES ON INTEGRATION, I

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Communicated June 3, 1948

The theory of integration, because of its central rôle in mathematical analysis and geometry, continues to afford opportunities for serious investigation. The need for extending and rounding out the classical studies of Riemann, Stieltjes and Lebesgue has stimulated considerable interest not only in new aspects of the theory but also in the simplification and perfection of the old. The present series of communications is intended to outline a treatment which, while exploiting fully the possibilities for simplification, will attain a high degree of generality. Among the many contributions to the mathematical literature which have provided material for our handling of the subject we wish to cite above all an important paper of Daniell.[1] In spite of the fact that the basic ideas in the

present discussion are the common property of mathematicians, some of our results appear to be novel. If they are, it is because we have chosen to introduce and exploit an adaptation of the concept of an "upper integral," making this the technical foundation of the whole theory.[2]

With Daniell, we assume an initially given *elementary integral* $E(f)$ which is defined for a non-void class $\mathfrak{E}$ of real functions f, called the *elementary functions*, with a fixed abstract set X as domain. Specifically, our basic postulates are the following slight modifications of those given by Daniell:

(1) $\mathfrak{E}$ is a non-void class of real functions f on an arbitrary non-void domain X, such that αf, $f + g$, and $|f|$ are in $\mathfrak{E}$ whenever α is a real number and f and g are both in $\mathfrak{E}$; and E is a real-valued function (or operation) defined over $\mathfrak{E}$, such that

$$E(\alpha f) = \alpha E(f),\ E(f + g) = E(f) + E(g),\ E(|f|) \geqq 0;$$

(2) if f and f_n are in $\mathfrak{E}$ and $|f| \leqq \sum_{n=1}^{\infty} |f_n|$, then

$$E(|f|) \leqq \sum_{n=1}^{\infty} E(|f_n|).$$

At a certain stage in the development, we shall introduce the further requirement that

(3) if f is in $\mathfrak{E}$, then $\min(1, f) = \frac{1}{2}(1 + f - |1 - f|)$ is in $\mathfrak{E}$.

Technically (1) means that $\mathfrak{E}$ is a vector lattice under its natural ordering and E is a positive linear functional on $\mathfrak{E}$. The rôles played by (2) and (3) will be brought out below.

In many important instances of the general theory, $\mathfrak{E}$ consists of the continuous real functions with compact nucleus[3] on a locally compact space X. Under these conditions (2) and (3) both follow from (1): (3) is evident, while (2) is established by constructing a non-negative elementary function g strictly positive on the nucleus of f and demonstrating the existence of an integer $m = m(\epsilon)$, $\epsilon > 0$, such that $|f| - \epsilon g \leqq \sum_{n=1}^{m} |f_n|$. By way of illustration we cite the Riemann integral on a bounded closed domain in n-space or on an n-dimensional manifold, and the Haar integral on a locally compact topological group.[4] Other important instances of the general theory are constructed from distributions of simple type given in advance on X: in X a class of subsets is distinguished and each distinguished set Y is assumed to bear a non-negative weight or measure, $\mu(Y)$; the real linear lattice combinations of the characteristic functions of distinguished sets are taken as the elementary functions; and the elementary integral is then determined in harmony with the requirement that $E(f) = \mu(Y)$ for the characteristic function f of any distinguished set Y. The detailed construction is not difficult once the appropriate tech-

niques have been provided for relating the data to the requirements of (1) and (2). We mention in particular that the given measure must be additive: if Y, Z, and $Y \cup Z$ are distinguished sets, and Y and Z are disjoint, then $\mu(Y \dot{\cup} Z) = \mu(Y) + \mu(Z)$. The theory of the Lebesgue-Stieltjes integral illustrates the general concepts which have just been described.

We shall consider the class $\mathfrak{G}$ of all extended-real functions defined on the domain X. The admission of $+\infty$ and $-\infty$ as functional values renders a little awkward the definition of such algebraic combinations of functions as $0 \cdot f, f + g$ and $f - g$. In the present context it is appropriate to let $0 \cdot f, f + g, f - g$ designate (ambiguously!) any function in $\mathfrak{G}$ which assumes the respective values $0 \cdot f(x), f(x) + g(x), f(x) - g(x)$ at every x where the latter quantities are defined. As we shall see, this ambiguity raises no serious obstacles in the sequel.

Over $\mathfrak{G}$ we define an extended-real function N by putting

$$(4) \quad N(f) = \inf \{\lambda;\ \lambda = \sum_{n=1}^{\infty} E(|f_n|),\ |f| \leqq \sum_{n=1}^{\infty} |f_n|, f_n \in \mathfrak{E}\}.$$

For non-negative functions, $N(f)$ has the properties of an upper integral; and for certain non-negative functions it coincides with the integral which is presently to be defined. Thus we already have in our possession the mathematical object upon which our interest is concentrated. The formal definition given in (4) is capable of an informal presentation which brings out clearly and rather simply its intuitive justification. Confining our attention to non-negative functions, we may regard (4) as the condensed description of a measuring process. We may think of $\mathfrak{E}$ as providing a stock of measuring rods, the non-negative elementary functions, by means of which the non-negative functions in $\mathfrak{G}$ are to be gauged. Each measuring rod has a magnitude given by its elementary integral. The basic measuring process consists in choosing from stock such an infinite sequence of measuring rods $f_n = |f_n| \geqq 0$ that by addition they combine to surpass $f = |f| \geqq 0$ in the sense indicated by the inequality $f \leqq \sum_{n=1}^{\infty} f_n$. The real number $\lambda = \sum_{n=1}^{\infty} E(f_n)$ obtained by adding together the magnitudes of the particular measuring rods thus employed is then accepted as an estimate, generally in excess, of the magnitude of f. Repetitions of this basic process furnish successively better estimates, convergent to the quantity $N(f)$, when suitable precautions are taken. It is, of course, conceivable that the basic process will fail to produce any real numbers as estimates of the magnitude of a particular function f—either because the inequality $f \leqq \sum_{n=1}^{\infty} f_n$ cannot be realized, or because it

implies the divergence of the series $\sum_{n=1} E(f_n)$. Under these circumstances the formal definition requires that we take $N(f) = +\infty$ in accordance with a well-known convention. The principal properties of the function N, all easily deduced from (4), can be listed as follows:

(5) $0 \leqq N(f) \leqq +\infty$;

(6) $N(\alpha f) = |\alpha| N(f)$ unless $\alpha = 0$ and $N(f) = +\infty$;

(7) $|f| \leqq \sum_{n=1}^{\infty} |f_n|$ implies $N(f) \leqq \sum_{n=1}^{\infty} N(f_n)$;

(8) $N(|f|) = N(f)$.

By specializing (7) we see that $N(f + g) \leqq N(f) + N(g)$, and that $N(f) \leqq N(g)$ whenever $|f| \leqq |g|$. The ambiguity of the expressions $\alpha f, f + g$ does not affect the truth of these relations; nor does (2) enter into their proofs. It is only in deriving the property

(9) when f is elementary, $N(f) = E(|f|)$

that (2) finds any direct application in our theory. Clearly (2) and (9) are equivalent assertions, so that whenever we use (9) in the sequel we also use (2) in an essential though implicit manner.

Henceforth we shall be concerned primarily with that part, $\mathfrak{F}$, of $\mathfrak{G}$ which is characterized by the inequality $N(f) < +\infty$. The expression $N(f - g)$ has in $\mathfrak{F}$ the properties of a pseudo-metric. Hence, if we identify functions f and g for which $N(f - g) = 0$, we can treat $\mathfrak{F}$ as a real normed vector-lattice with N as its norm-function. The detailed discussion involves attention to those functions f, called *null functions*, for which $N(f) = 0$. In this connection it is convenient to introduce also the following definitions: a subset of X is called a *null set* if its characteristic function is a null function; the phrase "*almost everywhere*" signifies "with the exception of the points of a certain null set." By the use of (7) it is easy to show that a function is a null function if and only if it vanishes almost everywhere; that any set covered by a countable family of null sets is a null set; and that every function in $\mathfrak{F}$ is finite almost everywhere. These and similar properties show that the null sets play here a rôle analogous to that played by the sets of measure zero in the Lebesgue theory. The identification of functions in $\mathfrak{F}$ is now seen to remove all ambiguity in the meaning of the expressions $\alpha f, f + g, f - g$ since every function in $\mathfrak{F}$ is finite almost everywhere. Once these more or less routine matters are disposed of, we can establish the most important single result[5] concerning $\mathfrak{F}$, namely:

(10) the normed vector space $\mathfrak{F}$ is complete (and hence a Banach space).

The proof will be sketched. Let $\{f_n\}$ be a Cauchy sequence in $\mathfrak{F}$. Without loss of generality we may suppose that f_n is everywhere finite (otherwise we could modify f_n on a null set so that the resulting function would be everywhere finite) and that $N(f_{n+1} - f_n) \leqq 2^{-n}$ (otherwise we could choose a subsequence which converges with the desired rapidity). The series $|f_1| + \sum_{n=1}^{\infty} |f_{n+1} - f_n|$ has the sum g in $\mathfrak{G}$. Since $N(g) \leqq N(f_1) + \sum_{n=1}^{\infty} N(f_{n+1} - f_n) = N(f_1) + \sum_{n=1}^{\infty} 2^{-n} \leqq N(f_1) + 1 < +\infty$ by (7), g is actually in $\mathfrak{F}$. Hence the series above converges almost everywhere, and so also do the series $f_1 + \sum_{n=1}^{\infty} (f_{n+1} - f_n)$ and the sequence $\{f_n\}$. Let f be any function in $\mathfrak{G}$ which is equal to the sum of the latter series or, equivalently, to $\lim_{n \to \infty} f_n$ wherever these quantities are defined: we may, for example, take $f = \limsup_{n \to \infty} f_n$ or $f = \liminf_{n \to \infty} f_n$. We then have $|f| \leqq g$, $N(f) \leqq N(g) < +\infty$, $f \in \mathfrak{F}$. Moreover $|f - f_k| \leqq \sum_{n=k}^{\infty} |f_{n+1} - f_n|$ and $N(f - f_k) \leqq \sum_{n=k}^{\infty} N(f_{n+1} - f_n) \leqq 2^{-k+1}$, so that the Cauchy sequence $\{f_n\}$ has f as its limit in $\mathfrak{F}$. This completes the proof.

On $\mathfrak{F}$ we now define the function F by putting $F(f) = N(f^+) - N(f^-)$ where $f^+ = \frac{1}{2}(|f|) + f) \geqq 0, f^- = \frac{1}{2}(|f| - f) \geqq 0$. Since $|f^+ - g^+| \leqq |f - g|$ and $|f^- - g^-| \leqq |f - g|$, this function is continuous in accordance with the inequalities $|F(f) - F(g)| \leqq |N(f^+) - N(g^+)| + |N(f^-) - N(g^-)| \leqq N(f^+ - g^+) + N(f^- - g^-) \leqq 2N(f - g)$. On $\mathfrak{E} \subset \mathfrak{F}$ we have $F(f) = E(f^+) - E(f^-) = E(f)$ by (9). Let $\mathfrak{L}$ be the closure of $\mathfrak{E}$ in $\mathfrak{F}$, and let L be the contraction of F to $\mathfrak{L}$. A function in $\mathfrak{L}$ is said to be *integrable*, and its *general integral* is taken to be $L(f) = N(f^+) - N(f^-)$. The integrable functions are the functions which can be approximated by elementary functions in the sense of the norm for $\mathfrak{F}$. On the other hand, the results of the preceding paragraph show that every integrable function f is equal almost everywhere to $\limsup_{n \to \infty} f_n$, where $\{f_n\}$ is a suitably chosen sequence of elementary functions. The following assertions concerning the general integral can now be justified:

(11) $\mathfrak{L}$ and L enjoy the properties assumed for $\mathfrak{E}$ and E, respectively, in (1), $\mathfrak{L}$ being a complete vector subspace of $\mathfrak{F}$ with norm $N(f) = L(|f|)$;

(12) the sum of a positive-term series of integrable functions is integrable if and only if the corresponding series of integrals converges (necessarily to the integral of the sum-function).

It is clear that (11) follows from (1) by simple continuity arguments. We observe that (12) may be regarded as a sharpened version of (2) formulated in terms of $\mathfrak{L}$ and L; and also that (12) is a generalized form of the

theorem of B. Levi in the Lebesgue theory. A proof of (12) will now be sketched. Let $f = \sum_{n=1}^{\infty} f_n$ where $f_n \geqq 0$ and $f_n \in \mathfrak{L}$. If $f \in \mathfrak{F}$, then $\sum_{n=1}^{m} L(f_n) = L(\sum_{n=1}^{m} f_n) = N(\sum_{n=1}^{m} f_n) \leqq N(f) < +\infty$ so that $\sum_{n=1}^{\infty} L(f_n)$ is a convergent positive-term series. On the other hand, if $\sigma = \sum_{n=1}^{\infty} L(f_n) < +\infty$, we have $N(f) \leqq \sum_{n=1}^{\infty} N(f_n) = \sum_{n=1}^{\infty} L(f_n) < +\infty$ so that $f \in \mathfrak{F}$. Moreover $N(f - \sum_{n=1}^{m} f_n) \leqq \sum_{n=m+1}^{\infty} N(f_n) = \sum_{n=m+1}^{\infty} L(f_n)$ so that $\sum_{n=1}^{m} f_n$ converges in $\mathfrak{L}$ to f and $L(\sum_{n=1}^{m} f_n)$ converges to $L(f)$, with the result that $L(f) = \sigma$. From (12) it is now possible to deduce other convergence theorems which correspond in our general theory to such standard results as the Lebesgue dominated-convergence theorem and the Fatou theorem in the Lebesgue theory. As the classical arguments apply without change to the present situation we do not need to go into detail.

Let Φ_m be the class of all positively homogeneous continuous real functions of m real variables $\lambda_1, \ldots, \lambda_m$. To Φ_m belong the functions $\lambda_1, \ldots, \lambda_m$ and all the linear lattice combinations which can be formed from them. On the other hand, it is known that on the compact set where $|\lambda_1| + \ldots + |\lambda_m| = 1$ any continuous function can be uniformly approximated by such combinations.[6] Hence if $\varphi \in \Phi_m$ we can find such a combination ψ_k that $|\varphi - \psi_k| \leqq 1/k$ on this set. It follows that $|\varphi - \psi_k| \leqq 1/k (|\lambda_1| + \ldots + |\lambda_m|)$. Since $\psi_k(f_1, \ldots, f_m)$ is integrable when $f_1, \ldots, f_m$ are, as we noted in (11), and since

$$N(\varphi(f_1, \ldots, f_m) - \psi_k(f_1, \ldots, f_m)) \leqq (N(f_1) + \ldots + N(f_m))/k \to 0$$

when $k \to \infty$, we conclude that $\varphi(f_1, \ldots, f_m)$ is integrable. The dominated-convergence theorem enables us to extend this result to the positively homogeneous Baire functions:

(13) if φ is a positively homogeneous real function of m real variables $\lambda_1, \ldots, \lambda_m$ whose contraction to the set $|\lambda_1| + \ldots + |\lambda_m| = 1$ is a bounded Baire function, and if $f_1, \ldots, f_m$ are integrable, then $\varphi(f_1, \ldots, f_m)$ is integrable.

In order to rid ourselves of the restriction to homogeneous functions, we now assume that (3) holds.[7] We find that (13) can be replaced by:

(14) if φ is a finite, not necessarily bounded Baire function of m real variables $\lambda_1, \ldots, \lambda_m$ such that $\varphi(0, \ldots, 0) = 0$, and if $f_1, \ldots, f_m, g$ are integrable functions such that $|\varphi(f_1, \ldots, f_m)| \leqq |g|$, then $\varphi(f_1, \ldots, f_m)$ is integrable.

Indeed, if the constant function everywhere equal to 1 is in $\mathfrak{E}$ or in $\mathfrak{L}$, we can also eliminate the condition $\varphi(0, \ldots, 0) = 0$. By further applica-

tion of the dominated-convergence theorem we can extend (13) and (14) to functions φ of infinitely many variables.

The principal processes for manipulating the general integral have now been justified. In our second note we shall review some of their applications and consequences.

[1] Daniell, P. J., "A General Form of Integral," *Ann. Math.*, **19**, 279–294 (1917–1918).

[2] Some remarks of H. Blumberg, *Am. Math. Monthly*, **53**, 189 (1946), on Lebesgue measure shed much light on the subject for me and started a train of thought culminating in the introduction of an "upper integral" here.

[3] The nucleus of a function is the closure of the set of points where it assumes non-zero values.

[4] Weil, A., *L'Intégration dans les Groupes Topologiques et ses Applications*, Paris, 1938, 34–38.

[5] This result is believed to be new.

[6] Stone, M. H., "The Generalized Weierstrass Approximation Theorem," *Math. Mag.* **21**, 167–183 (1948); see particularly §2, where Corollary 2 to Theorem 3 gives the relevant information.

[7] Other conditions leading to (14) have been investigated by Mr. H. Rubin, who, as a member of one of my classes, made many useful comments on the subject-matter of this whole paragraph.

ON THE DIFFERENTIAL EQUATIONS OF SLIP FLOW

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In a compressible medium, let the stress tensor be T^i_j, the specific internal energy ϵ, the specific entropy s, the specific volume σ. Then the *mean pressure* p_m and the *pressure* p are given by the definitions

$$p_m \equiv -\frac{1}{3} T^i_i, \qquad p \equiv -\left(\frac{\partial \epsilon}{\partial \sigma}\right)_s. \tag{1}$$

Let V_i be the velocity vector; then the *deformation* and *rotation* tensors d_{ij} and ω_{ij}, respectively, are given by the definitions

$$d_{ij} \equiv \frac{1}{2}(V_{i,j} + V_{j,i}), \qquad \omega_{ij} \equiv \frac{1}{2}(V_{i,j} - V_{j,i}). \tag{2}$$

If a secondary stress tensor W^i_j be given by the definition

$$W^i_j \equiv p\delta^i_j + T^i_j, \tag{3}$$

then the *dissipation function* Φ, given by the definition

$$\Phi = W^i_j d^j_i, \tag{4}$$

may be shown to be the rate of conversion of mechanical energy into thermal energy per unit volume.

A *compressible fluid* may be defined as a continuous medium obeying the following postulates:

I. There exist material constants μ_0, θ_0, and $\mathfrak{R}$ such that

$$\dim \mu_0 = \frac{\text{mass}}{(\text{length})\ (\text{time})};$$

$$\dim \theta_0 = \text{temperature};$$

$$\dim \mathfrak{R} = \frac{(\text{length})^2}{(\text{time})^2\ (\text{temperature})}.$$

II. $\Phi = \Phi(\mu_0, \theta_0, \mathfrak{R}; p_m; p, \theta; p_{,i}, \theta_{,i}, f_{i,j}, p_{,ij}, \theta_{,ij}; \ldots; f_{i_1,i_2} \ldots_{i_m}, p_{,i_1i_2}\ldots_{i_m}, \theta_{,i_1i_2}\ldots_{i_m}; d_{ij}, \omega_{ij}; c_{(1)}M_{(1)}, c_{(2)}M_{(2)}, \ldots, c_{(k)}M_{(k)}.)$

III. $W^i_j = 0$ if $V_{i,j} = 0$, $p_{,i} = 0$, $\theta_{,i} = 0$, $f_{i,j} = 0$.

In the above definition, θ is the temperature, f_i is the extraneous force per unit volume, $c_{(j)}$ is the molal concentration and $M_{(j)}$ is the molecular weight of the jth of the k phases of the various substances present, and m is an arbitrary positive integer.

If it be supposed that the function Φ is an analytic function of all its tensorial arguments (though not necessarily also of its scalar arguments), then it is possible to show that the definition just given leads to an expression for the stress tensor W^i_j of the general form

$$W^i_j = \sum_{r=1}^{\infty} \mu^r{}_{(r)} W^i_j, \tag{5}$$

where the quantity μ,

$$\mu = \mu_0 f\left(\frac{p}{p_m}, \frac{\theta}{\theta_0}, c_{(1)}M_{(1)}, c_{(2)}M_{(2)}, \ldots, c_{(k)}M_{(k)}\right), \tag{6}$$

the function f being a dimensionless function of its arguments, may be identified with the ordinary coefficient of viscosity. In the special case of an *isotropic* fluid it may then be shown that

$$_{(1)}\ W^i_j = \frac{\lambda}{\mu} d^k_k \delta^i_j + 2d^i_j, \tag{7}$$

$$_{(2)}\ W^i_j = \frac{\mathfrak{R}\theta}{p_m{}^3}\left[D_{(1)}p^{,k}p_{,k}\delta^i_j + 2C_{(1)}p^{,i}p_{,j}\right] + \frac{\mathfrak{R}}{p_m{}^2}\left[D_{(2)}p^{,k}\theta_{,k}\delta^i_j + C_{(2)}(p^{,i}\theta_{,j} + \theta^{,i}p_{,j})\right] + \frac{\mathfrak{R}}{p_m\theta}\left[D_{(3)}\theta^{,k}\theta_{,k}\delta^i_j + 2C_{(3)}\theta^{,i}\theta_{,j}\right] +$$

$$\frac{\mathfrak{R}\theta}{p_m{}^2}\left[D_{(4)}p^{,k}_{,k}\delta^i_j + 2C_{(4)}p^{,i}_{,j} + D_{(5)}f^k_{,k}\delta^i_j + C_{(5)}(f^i_{,j} + f^j_{,j})\right] +$$

$$\frac{\mathfrak{R}}{p_m}[D_{(6)}\theta^{,k}_{,k}\delta^i_j + 2C_{(6)}\theta^{,i}_{,j}] + \frac{1}{p_m}[F_{(1)}(d^k_k)^2\delta^i_j + F_{(2)}d^l_k d^k_l\delta^i_j + F_{(3)}\omega^l{}_k\omega^k{}_l\delta^i_j +$$
$$2E_{(1)}d^k_k\delta^i_j + 2E_{(2)}d^i_k d^k_j + 2E_{(3)}\omega^i{}_k\omega^k{}_j + E_{(4)}(d^{ik}\omega_{kj} + d_{jk}\omega^{ki})], \quad (8)$$

$${}_{(3)}W^i_j = \frac{\mathfrak{R}\theta}{p_m{}^4}[G_{(1)}d^k_k p^{,l}p_{,l}\delta^i_j + G_{(2)}d^{kl}p_{,l}p_{,k}\delta^i_j + G_{(3)}(d^{ik}p_{,k}p_{,j} +$$
$$d_{jk}p^{,k}p^{,i}) + G_{(4)}d^i_j p^{,k}p_{,k} + H_{(1)}(\omega^{ik}p_{,k}p_{,j} + \omega_{jk}p^{,k}p^{,i})] +$$
$$\frac{\mathfrak{R}}{p_m{}^3}[G_{(5)}d^k_k p^{,l}\theta_{,l}\delta^i_j + G_{(6)}d^{kl}p_{,k}\theta_{,l}\delta^i_j + G_{(7)}(d^{ik}p_{,k}\theta_{,j} + d_{jk}p^{,k}\theta^{,i}) +$$
$$G_{(8)}(d^{ik}\theta_{,k}p_{,j} + d_{jk}\theta^{,k}p^{,j}) + G_{(9)}d^i_j p^{,k}\theta_{,k} + H_2\omega^{kl}p_{,k}\theta_{,l}\delta^i_j +$$
$$H_{(3)}(\omega^{ik}p_{,k}\theta_{,j} + \omega_{jk}p^{,k}\theta^{,j}) + H_{(4)}(\omega^{ik}\theta_{,k}p_{,j} + \omega_{jk}\theta^{,k}p^{,j})] +$$
$$\frac{\mathfrak{R}}{p_m{}^2\theta}[G_{(10)}d^k_k\theta^{,k}\theta_{,k}\delta^i_j + G_{(11)}d^{kl}\theta_{,l}\theta_{,k}\delta^i_j + G_{(12)}(d^{ik}\theta_{,k}\theta_{,j} + d_{jk}\theta^{,k}\theta^{,i}) +$$
$$G_{(13)}d^i_j\theta^{,k}\theta_{,k} + H_{(5)}(\omega^{ik}\theta_{,k}\theta_{,j} + \omega_{jk}\theta^{,k}\theta^{,j})] + \frac{\mathfrak{R}\theta}{p_m{}^3}[G_{(14)}d^k_k p^{,l}_{,l}\delta^i_j +$$
$$G_{(15)}d^{kl}p_{,k\,l}\delta^i_j + G_{(16)}(d^{ik}p_{,kj} + d_{jk}p^{,ki}) + G_{(17)}d^k_k p^{,i}_{,j} + G_{(18)}d^i_j p^{,k}_{,k} +$$
$$H_{(6)}(\omega^{ik}p_{,kj} + \omega_{jk}p^{,ki}) + G_{(19)}d^k_k f^i_{,l}\delta^i_j + G_{(20)}d^{kl}f_{k,l}\delta^i_j + G_{(21)}(d^{ik}f_{k,j} +$$
$$d_{jk}f^{k,i}) + G_{(22)}(d^{ik}f_{j,k} + d_{jk}f^{i,k}) + G_{(23)}d^k_k(f^i_{,j} + f^{i}_{j}) + G_{(24)}d^i_j f^k_{,k} +$$
$$H_{(7)}\omega^{kl}f_{k,l}\delta^i_j + H_{(8)}(\omega^{ik}f_{k,j} + \omega_{jk}f^{k,i}) + H_{(9)}(\omega^{ik}f_{j,k} + \omega_{jk}f^{i,k})] +$$
$$\frac{\mathfrak{R}}{p_m{}^2}[G_{(25)}d^k_k\theta^{,l}_{,l}\delta^i_j + G_{(26)}d^{kl}\theta_{,k\,l}\delta^i_j + G_{(27)}(d^{ik}\theta_{,kj} + d_{jk}\theta^{,ki}) + G_{(28)}d^k_k\theta^{,i}_{,j} +$$
$$G_{(29)}d^i_j\theta^{,k}_{,k} + H_{(10)}(\omega^{ik}\theta_{,kj} + \omega_{jk}\theta^{,ki})] + \frac{1}{p_m{}^2}[K_{(1)}(d^k_k)^3\delta^i_j + K_{(2)}d^k_l d^i_k d^m_m\delta^i_j +$$
$$K_{(2)}d^k_l d^l_m d^m_k\delta^i_j + K_{(4)}d^k_k d^i_l d^l_j + K_{(5)}(d^k_k)^2 d^i_j + K_{(6)}d^i_k d^k_l d^l_j + L_{(1)}\omega^k{}_l d^l_m d^m_k\delta^i_j +$$
$$L_{(2)}(\omega^i{}_k d_{lj} + d^i_l\omega_{jk})d^{kl} + M_{(1)}\omega^k{}_l\omega^l{}_k d^m_m\delta^i_j + M_{(2)}\omega^k{}_l\omega^l{}_m d^m_k\delta^i_j +$$
$$M_{(3)}d^k_k\omega^{il}\omega_{lj} + M_{(4)}(\omega^i{}_k d_{lj} + d^i_l\omega_{jk})\omega^{kl} + N_{(1)}\omega^k{}_l\omega^l{}_m\omega^m{}_k\delta^i_j +$$
$$N_{(2)}(\omega^i{}_k\omega^k{}_l\omega^l{}_j + \omega_j{}^k\omega_k{}^l\omega_l{}^i)]. \quad (9)$$

Here λ is a function of the form (6), and the coefficients $C_{(i)}$, $D_{(i)}$, $E_{(i)}$, $F_{(i)}$, $G_{(i)}$, $H_{(i)}$, $K_{(i)}$, $L_{(i)}$, $M_{(i)}$, $N_{(i)}$ all are dimensionless functions of p/p_m, θ/θ_0, $c_{(1)}M_{(1)}$, $c_{(2)}M_{(2)}$, ..., $c_{(k)}M_{(k)}$. The expression for ${}_{(4)}W^i_j$ has also been written down, but since it contains several hundred terms it is too long to reproduce here.

Formula (7) is the Cauchy formula for the viscous stress in the ordinary theory of viscous compressible fluids. A special case of (8) has been derived by Burnett and Chapman-Cowling[1] from the kinetic theory of gases, following a method of formal solution of the Maxwell-Boltzmann equation due in principle to Hilbert and Enskog. Burnett's equations have recently been recommended by Tsien[2] and Schamberg[3] as describing aero-

dynamic phenomena encountered at altitudes of 100,000 to 300,000 feet in the earth's atmosphere. A portion of ${}_{(3)}W^i_j$ has been calculated from the kinetic theory by Chang and Uhlenbeck,[4] but their result is not available for comparison with (9).

It is interesting to notice that Burnett's expression for W^i_j contains but 7 moduli, while our equations (7) and (8) contain 21, so that to reduce our results to his not only must we discard ${}_{(3)}W^i_j$, ${}_{(4)}W^i_j$, ..., but also we must impose 14 special relations among the coefficients occurring in ${}_{(1)}W^i_j$ and ${}_{(2)}W^i_j$; they are

$$\left.\begin{aligned} &3\lambda + 2\mu = 0, \\ &3D_{(i)} + 2C_{(i)} = 0, \qquad i = 1, 2, \ldots 6, \\ &3F_{(i)} + 2E_{(i)} = 0, \qquad i = 1, 2, 3, \end{aligned}\right\} \tag{10}$$

$$C_{(1)} = C_{(4)} = -C_{(5)} = E_{(3)} = {}^1/_2 E_{(4)}. \tag{11}$$

The 10 reductions (10), of which the first is the classical Stokes relation, are necessary and sufficient conditions that

$$p = p_m. \tag{12}$$

The hypotheses of the kinetic theory, at least as employed in the present case, imply this equality of the two pressures at the outset. Since the pressure enters the various moduli λ, μ, $C_{(i)}$, $D_{(i)}$, $E_{(i)}$, $F_{(i)}$ only through the ratio p/p_m it follows that if the special relations (10) be adopted, then these moduli are independent of the pressure, a result familiar in the kinetic theory. It would be more accurate to say that the basic hypotheses of the kinetic theory (at least as employed by the authors mentioned) are such that it is plain from the outset that that theory is not sufficiently general to permit the dependence of the viscosity upon the pressure. The Stokes relation is not now generally accepted except for monatomic gases, nor does it seem probable that the remainder of (10) are correct. The physical meaning of the relations (11) is not apparent, and it does not seem likely they are correct.

The kinetic theory derivation is essentially the computation of successive approximations to a certain special type of solution of the Maxwell-Boltzmann equation, and there is no indication that the expression so derived at a given stage contains all the terms of a given order of magnitude; since the difficulties of calculation enormously increase for each stage, one is at present not able to compare the last terms retained even with the first neglected. The method of derivation presented here enables one to write down any number of terms, and hence suggests dimensionless characteristic numbers which determine the validity of a given approximation. One of these numbers, for example, is

$$\mathfrak{V} \equiv \frac{\mu\sqrt{d^i_j d^j_i}}{p_m}. \tag{13}$$

If $\mathfrak{V} \ll 1$ nô terms free of thermodynamic gradients need be retained except those whose coefficients are λ and μ, provided the coefficients $E_{(i)}$ and $F_{(i)}$ be not too large.

From the formula (8) may be deduced a rather startling consequence. In the case when the gas is in thermodynamic equilibrium and suffering no deformation whatever, but experiencing a uniform angular velocity ω about the z-axis, we obtain

$$W_{xx} = W_{yy} = -\frac{2\omega^2\mu^2}{p_m}(F_{(3)} + E_{(3)}), \qquad (14)$$

$$W_{zz} = -\frac{2\omega^2\mu^2}{p_m} F_{(3)}.$$

Thus a mass of fluid completely in equilibrium if set into rotation as a rigid body experiences a state of stress which depends in magnitude upon the viscosity of the fluid. Such an occurrence is most improbable. The elementary concept of a fluid would lead one to expect that the rate of dissipation of energy should be quite independent of the rotation, and that that only d_{ij} and not ω_{ij} should appear the basic postulate II. Indeed, in the present theory ω_{ij} was included only so as to obtain end formulae as general as those of kinetic theory. The result (14), however, which of course may be obtained also from the Burnett and Chapman-Cowling equations, suggests that perhaps there may have been an error in the kinetic theory analysis. The method used by Burnett and Chapman-Cowling does not appear to define uniquely the terms which occur at a given stage of the calculation, and it may be possible that in their result the terms involving ω_{ij} may be cancelled by terms from some higher order approximation which has not as yet been determined. This certain degree of arbitrariness may also explain the occurrence of the improbable relations (11) in Burnett's formula, since they might possibly be negated by as yet undiscovered higher order approximations. These remarks are offered only as suggestions. I have not repeated the kinetic theory calculations, which seem formidable. In any case, if ω_{ij} does not appear in Φ at all the end formulae are very greatly simplified, for then

$$F_{(3)} = E_{(3)} = E_{(4)} = H_{(1)} = L_{(1)} = M_{(1)} = N_{(1)} = 0. \qquad (15)$$

The analysis given here may easily be generalized so as to include incompressible fluids, for which the definition (1) for the pressure fails, and the pressure must be regarded as a primitive variable.

The preceding considerations are extracted from "A New Definition of a Fluid," shortly to be submitted to the *Journal of Mathematics and Physics*.

* This investigation was carried out under project ONR 45-47 from the Office of Naval Research to the Naval Ordnance Laboratory.

[1] Chapman, S., and Cowling, T. G., *The Mathematical Theory of Non-Uniform Gases*, Cambridge, 1939, see § 15.3 and § 15.4.

[2] Tsien, H. S., "Superaerodynamics, Mechanics of Rarefied Gases," *J. Aero. Sci.*, **13**, 653–664 (1946).

[3] Schamberg, R., *The Fundamental Differential Equations and the Boundary Conditions for High Speed Slip-Flow, and Their Application to Several Specific Problems*, thesis, Calif. Inst. Tech., 1947

[4] Chang, C. S. Wang, and Uhlenbeck, G. E., *On the Transport Phenomena in Rarified Gases*, Applied Physics Lab. report No. APL/JHU CM-443, 1948.

ERRATA

In the article, "Multiply Valued Harmonic Functions. Green's Theorem," by G. C. Evans, these PROCEEDINGS, **33**, 270–275 (1947), Lemma 1, line 3, replace "*each*" by "$\mu(e)$;" and Lemma 2, line 3, replace "*lower semi-continuous*" by "*its limit inferior for approach from T*."

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Lewis, Warren Kendall, 1938 (4), Massachusetts Institute of Technology, Cambridge 39, Mass.

Lind, Samuel Colville, 1930 (5), School of Chemistry, University of Minnesota, Minneapolis 14, Minn.

Link, Karl Paul, 1946 (9), Department of Biochemistry, Agricultural Experiment Station, University of Wisconsin, Madison, Wis.

Linton, Ralph, 1945 (11), Institute of Human Relations, 333 Cedar Street, New Haven 11, Conn.

Little, Clarence Cook, 1945 (10), Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Me.

Loeb, Leo, 1937 (10), 40 Crestwood Drive, St. Louis 5, Mo.

Loeb, Robert Frederick, 1946 (9), College of Physicians and Surgeons, 620 West 168th Street, New York 32, N. Y.

Long, Cyril Norman Hugh, 1948 (9), Yale University School of Medicine, 333 Cedar Street, New Haven 11, Conn.

Long, Esmond Ray, 1946 (10), Henry Phipps Institute, 7th and Lombard Streets, Philadelphia 47, Pa.

Longcope, Warfield Theobald, 1943 (10), Cornhill Farm, Lee, Mass.

Longsworth, Lewis Gibson, 1947 (5), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.

Longwell, Chester Ray, 1935 (6), Kirtland Hall, Yale University, New Haven, Conn.

Loomis, Alfred Lee, 1941 (4), The Loomis Laboratory, Room 2420, 14 Wall St., New York 5, N. Y.

Lowie, Robert Harry, 1931 (11), University of California, Berkeley 4, Calif.

Lyman, Theodore, 1917 (3), Research Laboratory of Physics, Harvard University, Cambridge 38, Mass.

McClintock, Barbara, 1944 (7), Carnegie Institution, Cold Spring Harbor, Long Island, N. Y.

McCollum, Elmer Verner, 1920 (9), Gilman Hall, Johns Hopkins University, Baltimore 18, Md.

McMillan, Edwin Mattison, 1947 (3), Radiation Laboratory, University of California, Berkeley 4, Calif.

McShane, Edward James, 1948 (1), School of Mathematics, University of Virginia, Charlottesville, Va.

MacInnes, Duncan Arthur, 1937 (5), Rockefeller Institute for Medical Reseach, 66th Street and York Avenue, New York 21, N. Y.

MacNider, William deBerniere, 1938 (9), University of North Carolina, Chapel Hill, N. C.

Macelwane, James Bernard, S.J., 1944 (6), 221 North Grand Boulevard, St. Louis 3, Mo.

Mangelsdorf, Paul Christoph, 1945 (7), Botanical Museum, Harvard University, Cambridge 38, Mass.

Marshall, Eli Kennerly, Jr., 1943 (9), School of Medicine, Johns Hopkins University, 710 North Washington Street, Baltimore 5, Md.

Marvel, Carl Shipp, 1938 (5), Department of Chemistry, University of Illinois, Urbana, Ill.

Mason, Max, 1923 (3), California Institute of Technology, Pasadena 4, Calif.

Mayer, Joseph Edward, 1946 (5), Department of Chemistry, University of Chicago, Chicago 37, Ill.

Maynard, Leonard Amby, 1944 (9), School of Nutrition, Cornell University, Ithaca, N. Y.

Mead, Warren Judson, 1939 (6), Massachusetts Institute of Technology, Cambridge 39, Mass.

Meek, Walter Joseph, 1947 (9), Department of Physiology, University of Wisconsin, Madison 6, Wis.

Mendenhall Walter Curran, 1932 (6), 9 East Lenox Street, Chevy Chase 15, Md.

Menzel, Donald Howard, 1948 (2), Harvard College Observatory, Cambridge 38, Mass.

Merica, Paul Dyer, 1942 (4), 67 Wall Street, New York 5, N. Y.

Merrill, Elmer Drew, 1923 (7), Arnold Arboretum, Jamaica Plain, Mass.

Merrill, Paul Willard, 1929 (2), Mount Wilson Observatory, Pasadena 4, Calif.

Metz, Charles William, 1948 (8), University of Pennsylvania, Zoological Laboratory, 38th Street and Woodland Avenue, Philadelphia 4, Pa.

Meyer, Karl Friederich, 1940 (10), George Williams Hooper Foundation, University of California Medical Center, San Francisco 22, Calif.

Michaelis, Leonor, 1943 (9), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.

Miles, Walter Richard, 1933 (12), Yale University School of Medicine, 333 Cedar Street, New Haven 11, Conn.

Miller, George Abram, 1921 (1), 1203 West Illinois Street, Urbana, Ill.

Millikan, Robert Andrews, 1915 (3), California Institute of Technology, Pasadena 4, Calif.

Minot, George Richards, 1937 (10), Thorndike Memorial Laboratory, Boston City Hospital, Boston 18, Mass.

Mitchell, Samuel Alfred, 1933 (2), Leander McCormick Observatory, University Station, Charlottesville, Va.

Moore, Carl Richard, 1944 (8), University of Chicago, Chicago 37, Ill.

Moore, Joseph Haines, 1931 (2), 6138 Swainland Road, Oakland 11, Calif.

Moore, Robert Lee, 1931 (1), University of Texas, Austin 12, Tex.

Morse, Harold **Marston,** 1932 (1), The Institute for Advanced Study, Princeton, N. J.

Moulton, Forest Ray, 1910 (2), 1515 Massachusetts Avenue, N. W., Washington 5, D. C.

Mueller, John **Howard,** 1945 (10), Harvard Medical School, 25 Shattuck Street, Boston 15, Mass.

Muller, Hermann Joseph, 1931 (8), Science Hall 101, Indiana University, Bloomington, Ind.

Mulliken, Robert Sanderson, 1936 (3), University of Chicago, Chicago 37, Ill.

Murnaghan, Francis Dominic, 1942 (1), Johns Hopkins University, Baltimore 18, Md.

Murphy, James Bumgardner, 1940 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.

Nicholson, Seth Barnes, 1937 (2), Mount Wilson Observatory, Pasadena 4, Calif.

Northrop, John Howard, 1934 (9), Rockefeller Institute for Medical Research, Princeton, N. J.

Novy, Frederick George, 1924 (10), 721 Forest Avenue, Ann Arbor, Mich.

Noyes, William Albert, Jr., 1943 (5), Department of Chemistry, University of Rochester, Rochester 3, N. Y.

Oncley, John Lawrence, 1947 (9), Department of Physical Chemistry, Harvard Medical School, Boston 15, Mass.

Onsager, Lars, 1947 (5), Sterling Chemistry Laboratory, Yale University, 225 Prospect Street, New Haven, Conn.

Opie, Eugene Lindsay, 1923 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.

Oppenheimer, J. Robert, 1941 (3), The Institute for Advanced Study, Princeton, N. J.

Osterhout, Winthrop John Vanleuven, 1919 (7), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.

Painter, Theophilus Shickel, 1938 (8), University of Texas, Austin 12, Tex.

Palache, Charles, 1934 (6), Harvard University, Cambridge 38, Mass.

Parker, George Howard, 1913 (8), Harvard Biological Laboratories, Divinity Avenue, Cambridge 38, Mass.

Patterson, John Thomas, 1941 (8), Department of Zoology, University of Texas, Austin 12, Tex.

Paul, John Rodman, 1945 (10), Yale University School of Medicine, 333 Cedar Street, New Haven 11, Conn.

Pauling, Linus, 1933 (5), Crellin Laboratory, California Institute of Technology, Pasadena 4, Calif.

Peters, John Punnett, 1947 (9), Department of Internal Medicine, Yale University School of Medicine, New Haven 11, Conn.

Pierce, George Washington, 1920 (3), Cruft Laboratory, Harvard University, Cambridge 38, Mass.

Piggot, Charles Snowden, 1946 (3), Committee on Geophysical Sciences, Research and Development Board, The Pentagon, Room 3D571, Washington 25, D. C.

Pillsbury, Walter Bowers, 1925 (12), University of Michigan, Ann Arbor, Mich.

Rabi, Isidor Isaac, 1940 (3), Department of Physics, Columbia University, New York 27, N. Y.

Reeside, John Bernard, Jr., 1945 (6), Box 177, Hyattsville, Md.

Reichelderfer, Francis Wilton, 1945 (3), Room 310, U. S. Weather Bureau, Washington 25, D. C.

Rhoades, Marcus Morton, 1946 (7), Department of Botany, University of Illinois, Urbana, Ill

Richards, Alfred Newton, 1927 (9), 291 Medical Laboratories, University of Pennsylvania, Philadelphia 4, Pa.

Richter, Curt Paul, 1948 (12), The Johns Hopkins Hospital, Baltimore 5, Md.

Riddle, Oscar, 1939 (8), Route 4, Plant City, Fla.

Ritt, Joseph Fels, 1933 (1), Columbia University, New York 27, N. Y.

Rivers, Thomas Milton, 1934 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.

Robbins, William Jacob, 1940 (7), New York Botanical Garden, Bronx Park (Fordham Station), New York 58, N. Y.

Robertson, Oswald Hope, 1943 (10), Department of Medicine, University of Chicago, Chicago 37, Ill.

Rodebush, Worth Huff, 1938 (5), University of Illinois, Urbana, Ill.

Romer, Alfred Sherwood, 1944 (8), Museum of Comparative Zoology, Oxford Street, Cambridge 38, Mass.

Rose, William Cumming, 1936 (9), University of Illinois, Urbana, Ill.

Ross, Frank Elmore, 1930 (2), Mount Wilson Observatory, Pasadena 4, Calif.

Rossby, Carl-Gustaf Arvid, 1943 (3), Institute of Meteorology, University of Chicago, Chicago 37, Ill. (Winter: Slottsvägen 131, Näsby, Stockholm, Sweden)

Rous, Francis Peyton, 1927 (10), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.

Rubey, William Walden, 1945 (6), U. S. Geological Survey, Washington 25, D. C.

Ruedemann, Rudolf, 1928 (6), 161 Dana Avenue, Albany 3, N. Y.

Russell, Henry Norris, 1918 (2), Princeton University, Princeton, N. J.

Sabin, Florence Rena, 1925 (10), 1333 East 10th Avenue, Denver 3, Colo.

Saunders, Frederick Albert, 1925 (3), South Hadley, Mass.

Sax, Karl, 1941 (7), Harvard University, Cambridge 38, Mass.

Scatchard, George, 1946 (5), Department of Chemistry, Massachusetts Institute of Technology, Cambridge 39, Mass.

Schlesinger, Hermann Irving, 1948 (5), Department of Chemistry, University of Chicago, Chicago 37, Ill.

Schmitt, Francis Otto, 1948 (8), Department of Biology, Massachusetts Institute of Technology, Cambridge 39, Mass.

Schultz, Adolph Hans, 1939 (11), Johns Hopkins Medical School, Baltimore 5, Md.

Seaborg, Glenn Theodore, 1948 (5), Department of Chemistry, University of California, Berkeley 4, Calif.

Seares, Frederick Hanley, 1919 (2), 351 Palmetto Drive, Pasadena 2, Calif.

Seashore, Carl Emil, 1922 (12), State University of Iowa, Iowa City, Iowa

Shaffer, Philip Anderson, 1928 (9), Washington University Medical School, St. Louis 10, Mo.

Shapley, Harlow, 1924 (2), Harvard College Observatory, Cambridge 38, Mass.

Sherman, Henry Clapp, 1933 (9), Columbia University, New York 27, N. Y.

Shope, Richard Edwin, 1940 (10), Box 323, Kingston, N. J.

Simpson, George Gaylord, 1941 (6), American Museum of Natural History, 77th Street and Central Park West, New York 24, N. Y.

Sinnott, Edmund Ware, 1936 (7), Osborn Botanical Laboratory, Yale University, New Haven, Conn.

Slater, John Clarke, 1932 (3), Massachusetts Institute of Technology, Cambridge 39, Mass.

Slepian, Joseph, 1941 (4), Westinghouse Electric Coropration, East Pittsburgh, Pa.

Slichter, Louis Byrne, 1944 (3), 1446 North Amalfi Drive, Pacific Palisades, Calif.

Slipher, Vesto Melvin, 1921 (2), Lowell Observatory, Flagstaff, Ariz.

Small, Lyndon Frederick, 1941 (5), National Institute of Health, Bethesda 14, Md.

Smith, Gilbert Morgan, 1948 (7), School of Biological Sciences, Stanford University, Stanford University, Calif.

Smith, Homer William, 1945 (9), 477 First Avenue, New York 16, N. Y.

Smith, Lee Irvin, 1944 (5), School of Chemistry, University of Minnesota, Minneapolis 14, Minn.

Smith, Paul Althaus, 1947 (1) Department of Mathematics, Columbia University, New York 27, N. Y.

Smith, Philip Edward, 1939 (8), College of Physicians and Surgeons, 630 West 168th Street, New York 32, N. Y.

Soderberg, Carl Richard, 1947 (4), Massachusetts Institute of Technology, Cambridge 39, Mass.

Sonneborn, Tracy Morton, 1946 (8), Department of Zoology, Indiana University, Bloomington, Ind.

Spier, Leslie, 1946 (11), P. O. Box 880, Santa Cruz, Calif.

Stadie, William Christopher, 1945 (9), 821 Maloney Clinic, 36th and Spruce Streets, Philadelphia 4, Pa.

Stadler, Lewis John, 1938 (7), University of Missouri, Columbia, Mo.

Stakman, Elvin Charles, 1934 (7), University Farm, St. Paul 8, Minn.

Stanley, Wendell Meredith, 1941 (9), Department of Biochemistry, University of California, Berkeley 4, Calif.

Stebbins, Joel, 1920 (2), Washburn Observatory, Madison 6, Wis. After Nov. 1, 1948: Lick Observatory, Mt. Hamilton, Calif.

Stern, Curt, 1948 (8), Department of Zoology, University of California, Berkeley 4, Calif.

Stern, Otto, 1945 (3), 759 Cragmont Avenue, Berkeley, Calif.

Stevens, Stanley Smith, 1946 (12), Department of Psychology, Harvard University, Cambridge 38, Mass.

Stewart, George Walter, 1938 (3), State University of Iowa, Iowa City, Iowa.

Stock, Chester, 1948 (6), Division of Geological Sciences, California Institute of Technology, Pasadena 4, Calif.

Stone, Calvin Perry, 1943 (12), Stanford University, Stanford University, Calif.

Stone, Marshall Harvey, 1938 (1), 313 Eckhart Hall, University of Chicago, Chicago 37, Ill.

Streeter, George Linus, 1931 (8), Johns Hopkins Medical School, 710 North Washington Street, Baltimore 5, Md.

Struve, Otto, 1937 (2), Yerkes Observatory, Williams Bay, Wis.

Sturtevant, Alfred Henry, 1930 (8), Califorinia Institute of Technology, Pasadena 4, Calif.

Suits, Chauncey Guy, 1946 (4), Research Laboratory, General Electric Company, Schenectady 5, N. Y.

Sumner, James Batcheller, 1948 (5), New York State College of Agriculture, Laboratory of Enzyme Chemistry, Cornell University, Ithaca, N. Y.

Sverdrup, Harald Ulrik, 1945 (3), Norsk Polarinstitutt, Observatoriegt. 1, Oslo, Norway

Swanton, John Reed, 1932 (11), 22 George Street, Newton 58, Mass.

Taliaferro, William Hay, 1940 (8), University of Chicago, Chicago 37, Ill.

Tate, John Torrence, 1942 (3), University of Minnesota, Minneapolis 14, Minn.

Teller, Edward, 1948 (3), Institute for Nuclear Studies, University of Chicago, Chicago 37, Ill.

Terman, Frederick Emmons, 1946 (4), School of Engineering, Stanford University, Stanford University, Calif.

Terman, Lewis, Madison, 1928 (12), 761 Dolores Street, Stanford University, Calif.

Thimann, Kenneth Vivian, 1948 (7), Harvard University, Biological Laboratories, 16 Divinity Avenue, Cambridge 38, Mass.

Thom, Charles, 1937 (7), 207 Grant Street, Port Jefferson, N. Y.

Thomas, Charles Allen, 1948 (4), Monsanto Chemical Company, 1700 South Second Street, St. Louis 4, Mo.

Thomas, Tracy Yerkes, 1941 (1), Swain Hall, Indiana University, Bloomington, Ind.

Thorndike, Edward Lee, 1917 (12), Teachers College, Columbia University, New York 27, N. Y.

Thurstone, Louis Leon, 1938 (12), University of Chicago, Chicago 37, Ill.

Timoshenko, Stephen Prokop, 1940 (4), Room 262, Engineering Building, Stanford University, Calif.

Tolman, Edward Chace, 1937 (12), Department of Psychology, University of California, Berkeley 4, Calif.

Tolman, Richard Chace, 1923 (5), California Institute of Technology, Pasadena 4, Calif.

Tozzer, Alfred Marston, 1942 (11), Peabody Museum, Harvard University, Cambridge 38, Mass.

Trumpler, Robert Julius, 1932 (2), Berkeley Astronomical Department, University of California, Berkeley 4, Calif.

Tuve, Merle Antony, 1946 (3), Department of Terrestrial Magnetism, Carnegie Institution of Washington, 5241 Broad Branch Road, N. W., Washington 15, D. C.

Tyzzer, Ernest Edward, 1942 (10), 175 Water Street, Wakefield, Mass.

Urey, Harold Clayton, 1935 (5), Institute for Nuclear Studies, University of Chicago, Chicago 37, Ill.

Vandiver, Harry Shultz, 1934 (1), Department of Applied Mathematics, University of Texas, Austin 12, Tex.

Van Niel, Cornelis Bernardus, 1945 (7), Hopkins Marine Station of Stanford University, Pacific Grove, Calif.

Van Slyke, Donald Dexter, 1921 (9), Rockefeller Institute for Medical Research, 66th Street and York Avenue, New York 21, N. Y.

Van Vleck, John Hasbrouck, 1935 (3), Harvard University, Cambridge 38, Mass.

Vaughan, Thomas Wayland, 1921 (6), 3333 P Street, N. W., Washington 7, D. C.

Veblen, Oswald, 1919 (1), The Institute for Advanced Study, Princeton, N. J.

Vickery, Hubert Bradford, 1943 (9), Connecticut Agricultural Experiment Station, New Haven 4, Conn.

von Karman, Theodore, 1938 (4), California Institute of Technology, Pasadena 4, Calif.

von Neumann, John, 1937 (1), The Institute for Advanced Study, Princeton, N. J.

Waksman, Selman Abraham, 1942 (7), Agricultural Experiment Station, New Brunswick, N. J.

Walker, John Charles, 1945 (7), 206 Horticulture Building, University of Wisconsin, Madison 6, Wis.

Walsh, Joseph Leonard, 1936 (1), Harvard University, Cambridge 38, Mass.

Webster, David Locke, 1923 (3), Physics Department, Room 385, Stanford University, Stanford University, Calif.

Weiss, Paul Alfred, 1947 (8), Department of Zoology, University of Chicago, Chicago 37, Ill.

Went, Frits Warmolt, 1947 (7), California Institute of Technology, Pasadena 4, Calif.

Werkman, Chester Hamlin, 1946 (9), Department of Bacteriology, Iowa State College, Ames, Iowa

Wetmore, Alexander, 1945 (8), Smithsonian Institution, Washington 25, D. C.

Wever, Ernest Glen, 1940 (12), Princeton University, Princeton, N. J.

Weyl, Claus Hugo Hermann, 1940 (1), The Institute for Advanced Study, Princeton, N. J.

Whipple, George Hoyt, 1929 (10), School of Medicine, University of Rochester, 260 Crittenden Boulevard, Rochester 7, N. Y.

Whitehead, John Boswell, 1932 (4), Johns Hopkins University, Baltimore 18, Md.

Whitney, Hassler, 1945 (1), Sudbury Road, Weston 93, Mass.

Whitney, Willis Rodney, 1917 (5), General Electric Company, Schenectady 5, N. Y.

Wigner, Eugene Paul, 1945 (3), 8 Ober Road, Princeton, N. J.

Williams, Robert R., 1945 (5), 297 Summit Avenue, Summit, N. J.
Williams, Roger John, 1946 (5), Biochemical Institute, University of Texas, Austin 12, Tex.
Willier, Benjamin Harrison, 1945 (8), Department of Biology, Johns Hopkins University, Baltimore 18, Md.
Willis, Bailey, 1920 (6), Box 1365, Stanford University, Calif.
Wilson, Edgar Bright, Jr., 1947 (5), Harvard University, Department of Chemistry, 12 Oxford Street, Cambridge 38, Mass.
Wilson, Edwin Bidwell, 1919 (3), Harvard School of Public Health, 695 Huntington Avenue, Boston 15, Mass.
Wilson, Robert Erastus, 1947 (4), 910 South Michigan Avenue, Chicago 80, Ill.
Wislocki, George Bernays, 1941 (8), Harvard Medical School, 25 Shattuck Street, Boston 15, Mass.
Wolbach, Simeon Burt, 1938 (10), Department of Pathology, Children's Hospital, 300 Longwood Avenue, Boston 15, Mass.
Wood, Robert Williams, 1912 (3), Johns Hopkins University, Baltimore 18, Md.
Woodring, Wendell Phillips, 1946 (6), United States Geological Survey, Washington 25, D. C.
Woodworth, Robert Sessions, 1921 (12), Columbia University, New York 27, N. Y.
Wright, Frederick Eugene, 1923 (6), 2134 Wyoming Avenue, N. W., Washington 8, D. C.
Wright, Sewall Green, 1934 (8), Department of Zoology, University of Chicago, Chicago 37, Ill.
Wright, William Hammond, 1922 (2), 60 North Keeble Avenue, San Jose 11, Calif.
Yerkes, Robert Mearns, 1923 (12), Yale University School of Medicine, 333 Cedar Street, New Haven, Conn.
Yost, Don Merlin Lee, 1944 (5), California Institute of Technology, Pasadena 4, Calif.
Zariski, Oscar, 1944 (1), Department of Mathematics, Harvard University, Cambridge 38, Mass.
Zworykin, Vladimir Kosma, 1943 (4), 103 Battle Road, Princeton, N. J.

Number of Members July 1, 1948: 430

MEMBERS EMERITI

Benedict, Francis Gano, 1914, Machiasport, Me.
Dewey, John, 1910, 1158 Fifth Avenue, New York 29, N. Y.
Dickson, Leonard Eugene, 1913, Route #2, Joliet, Ill.
Howard, Leland Ossian, 1916, Bureau of Entomology, U. S. Department, of Agriculture, Washington 25, D. C.
Stratton, George Malcolm, 1928, University of California, Berkeley 4, Calif.

FOREIGN ASSOCIATES

The letter in parentheses following the year of election indicates the field of scientific research in which the foreign associate was working at the time of his election, as follows:

(A) Mathematics
(B) Astronomy
(C) Physics
(D) Engineering
(E) Chemistry
(F) Geology and Paleontology
(G) Botany
(H) Zoology and Anatomy
(I) Physiology and Biochemistry
(J) Pathology and Bacteriology
(K) Anthropology and Psychology

Adrian, Edgar Douglas, 1941 (I), Trinity College, Cambridge, England

Alexandroff, Paul A., 1947 (A), Mathematical Institute of the Academy of Sciences of the U.S.S.R., Bolshaya Kalushskaya 19, Moscow, U.S.S.R.

Bailey, Sir Edward, 1944 (F), 19 Greenhill Gardens, Edinburgh 10, Scotland

Bartlett, Frederic Charles, 1947 (K), University of Cambridge, The Psychological Laboratory, Downing Place, Cambridge, England

Bjerknes, V. (F. K.), 1934 (F), The University, Oslo, Norway

Bohr, Niels, 1925 (C), The Institute for Advanced Study, Princeton, N. J. (U.S.A.)

Bordet, Jules, 1935 (I), Pasteur Institute, Rue du Remorqueur, 28, Brussels, Belgium

Bragg, Sir William **Lawrence,** 1945 (C), Cavendish Laboratory, The University, Cambridge, England

de Broglie, Prince Louis, 1948 (C), 94 Rue Perronet, Neuilly-sur-Seine, France

Caso, Alfonso, 1943 (K), Secretaria de Bienes Nacionales e Inspeccion Administrativa, Mexico, D. F.

Chapman, Sydney, 1946 (A), Letters: Queen's College, Oxford; Printed matter: The Mathematical Institute, Oxford, England

Dale, Sir Henry Hallett, 1940 (I), The Wellcome Trust, 28 Portman Square, London, W.1, England

Debye, Peter, 1931* (C), Baker Laboratory, Cornell University, Ithaca, N. Y. (U.S.A.)

Einstein, Albert, 1922† (C), The Institute for Advanced Study, Princeton, N. J. (U.S.A.)

Fisher, Ronald Aylmer, 1948 (H), Department of Genetics, University of Cambridge, 44 Storey's Way, Cambridge, England

Hadamard, Jacques, 1926 (A), 12, rue Emile Faguet, Paris XIV, France

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* Dr. Debye became a naturalized citizen in 1946 and was elected a member of the Academy in 1947.

† Dr. Einstein became a naturalized American citizen in 1941 and was elected a member of the Academy in 1942.

If a foreign associate becomes a member of the Academy his name is not counted in the limit of 50 foreign associates.

Hardy, Godfrey Harold, 1927 (A), Trinity College, Cambridge, England
Helland-Hansen, Björn, 1947 (F), Chr. Michelsens Institutt for Videnscap, Bergen, Norway
Hill, Archibald Vivian, 1941 (I), 16 Bishopwood Road, Highgate, London, N.6, England
Hill, James Peter, 1940 (H), Kanimbla, Dollis Avenue, London N.3, England
Houssay, Bernardo Alberto, 1940 (I), Viamonte 2790, Buenos Aires, Argentina
Janet, Pierre, 1938 (K), 54, rue de Varenne, Paris VII, France
Jeffreys, Harold, 1945 (B), St. John's College, Cambridge, England
Jones, Sir Harold Spencer, 1943 (B), Royal Observatory, Greenwich, London, S.E.10, England
Kapitza, Peter Leonidovich, 1946 (C), Institute for Physical Problems, Academy of Sciences of the U.S.S.R., Moscow, U.S.S.R.
Karrer, Paul, 1945 (E), University of Zurich, Zurich, Switzerland
Keith, Sir Arthur, 1941 (I, K), Buckston Browne Farm, Downe, Farnborough, Kent, England
Krogh, August, 1937 (H), The Laboratory, Söbredden, Gentofte, Denmark
Levi, Giuseppe, 1940 (H), Instituto di Anatomia Umana, Corso Massimo D'Azeglio, 52, Turin, Italy
Limm, Robert K. S., 1942 (I), Army Medical Administration, Municipal Government, Shanghai, China
Linderstrøm-Lang, Kaj Ulrik, 1947 (E), Chemical Division, Carlsberg Laboratory, Copenhagen, Denmark
Robinson, Sir Robert, 1934 (E), Dyson Perrins Laboratory, South Parks Road, Oxford, England
Ruzicka, Leopold, 1944 (E), Department of Organic Chemistry, Institute of Technology, Zurich, Switzerland
Sherrington, Sir Charles Scott, 1924 (I), Gonville and Caius College, Cambridge, England
Sommerfeld, Arnold, 1929 (C), Dunant-strasse 6, Munich 23, Germany
Southwell, Richard Vynne, 1943 (D), Imperial College of Science and Technology, South Kensington, London, S.W.7, England
Svedberg, The, 1945 (E), Fysikalisk-Kemiska Institutionen, University of Uppsala, Uppsala, Sweden
Taylor, Sir Geoffrey I., 1945 (A), Trinity College, Cambridge, England
Vallee-Poussin, C. de la, 1929 (A), University of Louvain, Louvain, Belgium
Van der Bijl, Hendrik Johannes, 1943 (D), 722 Escom House, Rissik Street, Johannesburg, South Africa
Vening Meinesz, Felix Andries, 1939 (F), Potgieterlaan 5, Amersfoort, Holland
Watson, D. M. S., 1938 (H), University College, Gower Street, London, W.C.1, England
Wieland, Heinrich, 1932 (E), Sophienstrasse 9, Munich 2 NW, Germany

Number of Foreign Associates July 1, 1948: 41.

SECTIONS

(1) *Mathematics*—26 members

von Neumann, John, *Chairman* (1949)
Albert, A. A.
Alexander, J. W.
Bell, E. T.
Bliss, G. A.
Coble, A. B.
Douglas, Jesse
Eisenhart, L. P.
Evans, G. C.
Kasner, Edward
Lefschetz, Solomon
McShane, E. J.
Miller, G. A.
Moore, R. L.
Morse, Marston
Murnaghan, F. D.
Ritt, J. F.
Smith, P. A.
Stone, M. H.
Thomas, T. Y.
Vandiver, H. S.
Veblen, Oswald
Walsh, J. L.
Weyl, Hermann
Whitney, Hassler
Zariski, Oscar

(2) *Astronomy*—26 members

Mitchell, S. A., *Chairman* (1950)
Abbot, C. G.
Adams, W. S.
Aitken, R. G.
Anderson, J. A.
Babcock, H. D.
Bowen, I. S.
Fleming, J. A.
Hubble, E. P.
Joy, A. H.
King, A. S.
Leuschner, A. O.
Menzel, D. H.
Merrill, P. W.
Moore, J. H.
Moulton, F. R.
Nicholson, S. B.
Ross, F. E.
Russell, H. N.
Seares, F. H.
Shapley, Harlow
Slipher, V. M.
Stebbins, Joel
Struve, Otto
Trumpler, R. J.
Wright, W. H.

(3) *Physics*—60 members

Beams, J. W., *Chairman* (1951)
Allison, S. K.
Alvarez, L. W.
Anderson, C. D.
Bacher, R. F.
Bainbridge, K. T.
Berkner, L. V.
Bethe, H. A.
Birge, R. T.
Bjerknes, Jacob
Bloch, Felix
Breit, Gregory
Bridgman, P. W.
Coblentz, W. W.
Compton, A. H.
Compton, K. T.
Condon, E. U.
Coolidge, W. D.
Crew, Henry
Davis, Bergen
Davisson, C. J.
Dempster, A. J.
DuBridge, L. A.
Dunning, J. R.
Einstein, Albert
Epstein, P. S.
Fermi, Enrico
Franck, James
Goudsmit, S. A.
Houston, W. V.
Hull, A. W.
Ives, H. E.
Kemble, E. C.
Lauritsen, C. C.
Lawrence, E. O.
Lyman, Theodore
McMillan, E. M.
Mason, Max
Millikan, R. A.
Mulliken, R. S.
Oppenheimer, J. R.
Pierce, G. W.
Piggot, C. S.
Rabi, I. I.
Reichelderfer, F. W.
Rossby, C.-G.
Saunders, F. A.
Slater, J. C.
Slichter, L. B.
Stern, Otto
Stewart, G. W.
Sverdrup, H. U.
Tate, J. T.
Teller, Edward
Tuve, M. A.
Van Vleck, J. H.
Webster, D. L.
Wigner, E. P.
Wilson, Edwin B.
Wood, R. W.

(4) *Engineering*—35 members

Dryden, H. L., *Chairman* (1950)	Foote, P. D.	Loomis, A. L.
Adams, C. A.	Gilliland, E. R.	Merica, P. D.
Briggs, L. J.	Herty, C. H., Jr.	Slepian, Joseph
Buckley, O. E.	Hoover, Herbert	Soderberg, C. R.
Bush, Vannevar	Hovgaard, William	Suits, C. G.
Cochrane, E. L.	Hunsaker, J. C.	Terman, F. E.
Cottrell, F. G.	Jeffries, Zay	Thomas, C. A.
Curme, G. O., Jr.	Jewett, F. B.	Timoshenko, Stephen
Dunn, Gano	Kelly, M. J.	von Karman, T.
Durand, W. F.	Kettering, C. F.	Whitehead, J. B.
Fletcher, Harvey	Lewis, G. W.	Wilson, R. E.
	Lewis, W. K.	Zworykin, V. K.

(5) *Chemistry*—58 members

Latimer, W. M., *Chairman* (1950)	Gilman, Henry	Marvel, C. S.
Adams, L. H.	Hammett, L. P.	Mayer, J. E.
Adams, Roger	Harkins, W. D.	Noyes, W. A., Jr.
Adkins, Homer	Hildebrand, J. H.	Onsager, Lars
Bachmann, W. E.	Hudson, C. S.	Pauling, Linus
Bancroft, W. D.	Hulett, G. A.	Rodebush, W. H.
Bartlett, P. D.	Ipatieff, V. N.	Scatchard, George
Baxter, G. P.	Jacobs, W. A.	Schlesinger, H. I.
Bogert, M. T.	Johnson, J. R.	Seaborg, G. T.
Bolton, E. K.	Keyes, F. G.	Small, L. F.
Conant, J. B.	Kharasch, M. S.	Smith, L. I.
Cope, A. C.	Kirkwood, J. G.	Sumner, J. B.
Daniels, Farrington	Kistiakowsky, G. B.	Tolman, R. C.
Debye, Peter	Kraus, C. A.	Urey, H. C.
Eyring, Henry	La Mer, V. K.	Whitney, W. R.
Fieser, L. F.	Lamb, A. B.	Williams, Robert R.
Folkers, Karl	Langmuir, Irving	Williams, Roger J.
Fuson, R. C.	Lind, S. C.	Wilson, E. Bright, Jr.
Giauque, W. F.	Longsworth, L. G.	Yost, D. M.
	MacInnes, D. A.	

(6) *Geology*—38 members

Rubey, W. W., *Chairman* (1951)	Buddington, A. F.	Gregory, W. K.
Allen, E. T.	Byerly, Perry	Gutenberg, Beno
Berkey, C. P.	Chaney, R. W.	Hewett, D. F.
Blackwelder, Eliot	Cross, Whitman	Kelley, W. P.
Bowen, N. L.	Daly, R. A.	Knopf, Adolph
Bowman, Isaiah	Day, A. L.	Larsen, E. S., Jr.
Bradley, W. H.	Dunbar, C. O.	Lawson, A. C.
Bucher, W. H.	Ewing, Maurice	Leith, C. K.
	Gilluly, James	Longwell, C. R.

Macelwane, J. B.
Mead, W. J.
Mendenhall, W. C.
Palache, Charles
Reeside, J. B., Jr.
Ruedemann, Rudolf
Simpson, G. G.
Stock, Chester
Vaughan, T. W.
Willis, Bailey
Woodring, W. P.
Wright, F. E.

(7) *Botany*—35 members

Cleland, R. E., *Chairman* (1950)
Allen, C. E.
Babcock, E. B.
Bailey, I. W.
Bailey, L. H.
Beadle, G. W.
Blakeslee, A. F.
Brink, R. A.
Campbell, D. H.
Chandler, W. H.
Couch, J. N.
Dodge, B. O.
Duggar, B. M.
Fernald, M. L.
Fred, E. B.
Hoagland, D. R.
Jones, D. F.
Kunkel, L. O.
McClintock, Barbara
Mangelsdorf, P. C.
Merrill, E. D.
Osterhout, W. J. V.
Rhoades, M. M.
Robbins, W. J.
Sax, Karl
Sinnott, E. W.
Smith, Gilbert M.
Stadler, L. J.
Stakman, E. C.
Thimann, K. V.
Thom, Charles
Van Niel, C. B.
Waksman, S. A.
Walker, J. C.
Went, F. W.

(8) *Zoology and Anatomy*—38 members

Wright, Sewall, *Chairman*, (1949)
Bigelow, H. B.
Castle, W. E.
Child, C. M.
Conklin, E. G.
Corner, G. W.
Danforth, C. H.
Demerec, Milislav
Detwiler, S. R.
Dobzhansky, Theodosius
Dunn, L. C.
Goldschmidt, R. B.
Harrison, R. G.
Hartman, C. G.
Harvey, E. N.
Herrick, C. J.
Hisaw, F. L.
Jacobs, M. H.
Lewis, W. H.
Metz, C. W.
Moore, Carl R.
Muller, H. J.
Painter, T. S.
Parker, G. H.
Patterson, J. T.
Riddle, Oscar
Romer, A. S.
Schmitt, F. O.
Smith, P. E.
Sonneborn, T. M.
Stern, Curt
Streeter, G. L.
Sturtevant, A. H.
Taliaferro, W. H.
Weiss, Paul
Wetmore, Alexander
Willier, B. H.
Wislocki, G. B.

(9) *Physiology and Biochemistry*—45 members

Doisy, E. A., *Chairman* (1951)
Anderson, R. J.
Ball, E. G.
Bard, Philip
Bronk, D. W.
Carlson, A. J.
Clark, W. M.
Clarke, H. T.
Cohn, E. J.
Cori, Carl F.
Cori, Gerty T.
Davis, Hallowell
DuBois, E. F.
du Vigneaud, Vincent
Elvehjem, C. A.
Erlanger, Joseph
Evans, H. M.
Fenn, W. O.
Forbes, Alexander
Gasser, H. S.
Hart, E. B.
Hartline, H. K.
Hastings, A. B.
Link, K. P.
Loeb, R. F.
Long, C. N. H.
McCollum, E. V.
MacNider, W. deB.
Marshall, E. K., Jr.
Maynard, L. A.
Meek, W. J.
Michaelis, Leonor

Northrop, J. H.
Oncley, J. L.
Peters, J. P.
Richards, A. N.
Rose, W. C.
Shaffer, P. A.
Sherman, H. C.
Smith, Homer W.
Stadie, W. C.
Stanley, W. M.
Van Slyke, D. D.
Vickery, H. B.
Werkman, C. H.

(10) *Pathology and Bacteriology*—37 members

Dochez, A. R., *Chairman* (1951)
Addis, Thomas
Armstrong, Charles
Avery, O. T.
Blake, F. G.
Blalock, Alfred
Cannon, P. R.
Castle, W. B.
Cole, Rufus
Dubos, R. J.
Francis, Thomas, Jr.
Gamble, J. L.
Goodpasture, E. W.
Graham, E. A.
Heidelberger, Michael
Hektoen, Ludvig
Horsfall, F. L., Jr.
Kelser, R. A.
Little, C. C.
Loeb, Leo
Long, E. R.
Longcope, W. T.
Meyer, K. F.
Minot, G. R.
Mueller, J. Howard
Murphy, J. B.
Novy, F. G.
Opie, E. L.
Paul, John R.
Rivers, T. M.
Robertson, O. H.
Rous, Peyton
Sabin, Florence R
Shope, R. E.
Tyzzer, E. E.
Whipple, G. H.
Wolbach, S. B.

(11) *Anthropology*—9 members

Linton, Ralph, *Chairman* (1951)
Hooton, E. A.
Kidder, A. V.
Kroeber, A. L.
Lowie, R. H.
Schultz, A. H.
Spier, Leslie
Swanton, J. R.
Tozzer, A. M.

(12) *Psychology*—23 members

Boring, E. G., *Chairman* (1950)
Angell, J. R.
Carmichael, Leonard
Gesell, Arnold
Graham, C. H.
Hilgard, E. R.
Hull, C. L.
Hunter, W. S.
Köhler, Wolfgang
Lashley, K. S.
Miles, W. R.
Pillsbury, W. B.
Richter, C. P.
Seashore, C. E.
Stevens, S. S.
Stone, C. P.
Terman, L. M.
Thorndike, E. L.
Thurstone, L. L.
Tolman, E. C.
Wever, E. G.
Woodworth, R. S.
Yerkes, R. M.

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